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*Butterfield, T. G., and Starkweather, G. A.: J. Philadelphia Gen. Hosp. 2:6 (Jan.) 1951.

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South African Medical Journal
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CONGENITAL HYPOPLASIA OF THE GALL BLADDER WITH CYST-HEPATIC DUCT

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Department of Pathology, University of Cape Town

True congenital hypoplasia of the gall bladder unassociated with atresia of the extra-hepatic biliary system appears to be an extremely rare condition. It is not mentioned in the standard textbooks of anatomy and surgery as a rule, or is dismissed without reference to the literature. The *Index Medicus* contains no reference to the condition between 1920 and 1949.

In association with atresia or absence of the biliary tract, the gall bladder may either be represented by a strand of fibrous tissue or be absent, and the depression on the under surface of the right lobe of the liver may also be absent. There are a few cases, however, in which the biliary system is described as being normal apart from either absence or atresia of the gall bladder. Roth (1868) describes a case in which the gall bladder was represented by a fibrous band with a small mucus-filled cyst on either side of it—these did not communicate with the rest of the biliary system, which was normal. In Lockhardt's case (1927) '. . . there was a little pouch of the common bile duct suggestive of an arrested attempt to produce the gall bladder in the normal position'. The gall bladder depression was absent, and the rest of the biliary system was normal. However, no further details and no measurements are given. Gross (1936) in his review of anomalies of the gall bladder mentions several other references which were not available for study. He himself apparently had not seen a case of hypoplasia without either obliteration or inflammatory scarring of the bile ducts, and he rightly emphasizes that differentiation must be clearly established in each case between congenital hypoplasia and post-inflammatory shrinkage. Indeed, he goes so far as to say that he has never seen a case in which he was satisfied that the reduction in size was not due to inflammatory causes.

Cyst-hepatic ducts are a recognized hazard in cholecystectomy and may or may not be associated with other accessory ducts. The case reported below had congenital gall bladder hypoplasia with a cyst-hepatic duct, and illustrates the possible operative hazards associated with the latter.

REPORT OF A CASE

A 74-year-old Cape Coloured female was admitted in a state of coma to a medical ward in Groote Schuur Hospital on 16 March 1950. She had been suffering from progressive weakness for six weeks and had been confined to bed for one week. For three days there had been increasing mental confusion, and for two days she had had rigors associated with pain in the left side.

Two years before the patient had had an attack of jaundice associated with bile and sugar in the urine—no further details could be obtained. Her health in the interim had been good. The practitioner who arranged for her admission stated that he had found bile in her urine two weeks before admission, and at the time when the rigors commenced the patient had had polydypsia, the urine contained a trace of sugar but no bile and he had treated her with Sulphadiazine.

On examination the obese, slightly jaundiced patient could be aroused only by supra-orbital pressure. There was a moderate degree of hyperpnoea, but signs of ketosis were thought to be far too slight to account for the depth of coma. Neck rigidity was present; Kernig's and Brudzinski's signs were positive. The only physical findings apart from this were consolidation of both lung bases and an ill-defined mass in the left loin.

Blood. Red blood cells 3.7 million per c.mm.; packed cell volume, 33%; erythrocyte sedimentation rate, 55 mm. in one hour; white blood cells, 17,640 per c.mm. with 88% of polymorphs.

Urine. Specific gravity 1015; albumin, —; sugar, brick-red reaction; acetone, +; aceto-acetic acid, —. Spun deposit: moderate numbers of pus cells and epithelial cells.

The patient was treated with intravenous fluids, Penicillin and Insulin, but died in peripheral collapse within 24 hours of admission.

AUTOPSY

Autopsy was performed 10 hours after death. The body was that of an obese elderly woman showing slight jaundice of the sclerae but not of the skin.

The heart was normal apart from atheroma of the anterior mitral valve cusp and coronary arteries. The great vessels showed atherosclerosis. The lungs were oedematous and showed acute bronchitis and multiple septic infarcts with overlying fibrinous pleurisy, the largest 2 cm. in diameter. The gastro-intestinal tract was normal apart from the duodenum, which will be described later. The spleen showed an 'acute splenic tumour'. The left kidney was much enlarged, showing an extensive acute pyelonephritis at the upper pole. The right kidney showed a large subcapsular abscess into which haemorrhage had occurred. The lower limit of the abscess was formed by

adherence of the capsule over a typical flat pyelonephritic scar involving the lower one-third of the kidney. On section, there was an acute pyelonephritis of the upper two-thirds of the organ with a necrotizing papillitis and septic thrombosis of several of the renal vein tributaries. Culture from the necrotic papillae gave a growth of *Aerobacter aërogenes* and of a coagulase positive *Micrococcus pyogenes aureus*. The brain showed an early basal purulent meningitis and a Gram-stained smear contained numerous pus cells and Gram-positive cocci in clusters.

The Biliary System. The external surface of the liver appeared normal. There were dense adhesions in the region of the porta hepatis lifting the second part of the duodenum upwards and to the right. When these had been dissected away it was seen that the gall bladder was a tiny structure, half buried in the right lobe of the liver and measuring 1.8 by 0.7 cm. The lumen was filled by a large facetted calculus, and at the fundus of the gall bladder there was a cyst-hepatic duct which admitted a large probe. The mucosa of the gall bladder showed no abnormality to the naked eye, and the wall varied in thickness from 0.1 to 0.4 cm. The cystic duct was short (1.0 cm.) and wide (0.5 cm.) and the normal spiral valve was present (Fig. 1). Beyond the tip of the gall bladder there was no gall bladder depression.

The common bile duct and the main hepatic ducts were dilated, and the wall of the common bile duct was thickened. Though the lumen of the common bile duct was 0.8 cm. in diameter at its widest point, no calculus was found, and no other cause of obstruction demonstrated. On tracing the duct down to the duodenum, no duodenal papilla was found. Instead, the common bile duct and the pancreatic duct, which was normal, opened just below the mouth of a large diverticulum. The diverticulum had a mouth 2 cm. wide, was 2 cm. deep, and the base was 3 cm. wide, resting on the pancreas and separated from it by only a thin layer of connective tissue. The mucosa lining the diverticulum appeared normal.

On section of the liver, the parenchyma appeared normal except in relation to the cyst-hepatic duct, next to which a thrombosed portal vein could be seen, and this was the centre of a red infarct 4 cm. in diameter.

Microscopy confirmed the naked-eye findings. In addition, the kidneys showed an inter-capillary glomerulosclerosis. The lesion in the liver was seen to be an area of intense, localized passive congestion, i.e. the appearance of the so-called 'red infarct'. The thrombus in the portal vein was organized and the neighbouring bile duct showed old inflammatory scarring. The mucosa of the gall bladder was absent, the submucosa showed heavy round cell infiltration. There was some scarring of the muscle layer while beyond was a layer of scar tissue and some newly formed granulation tissue.

EMBRYOLOGY

During the fourth week of intra-uterine life the hepatic bud arises ventrally from the entodermal lining of the gut, then extends into, and splits the ventral mesentery. The distal part of the bud divides into numerous branching cell columns which soon become multiple hepatic ducts with proliferating liver cells beyond. These numerous ducts later fuse to form the left and right hepatic ducts.

By the beginning of the fifth week, at the point of fusion of the hepatic ducts, the gall bladder diverticulum appears, rapidly elongates and differentiates into the cystic duct and gall bladder. During the second lunar month of foetal life the gall bladder becomes buried in the right lobe of the liver and, in common with the cystic, hepatic and common bile ducts, its lumen becomes obliterated by epithelial proliferation. Recanalization follows and is usually complete by the twelfth week, at which time the liver commences to secrete bile, but it is only during the fourth month that the spiral ridge, which becomes the spiral valve of Heister, appears.

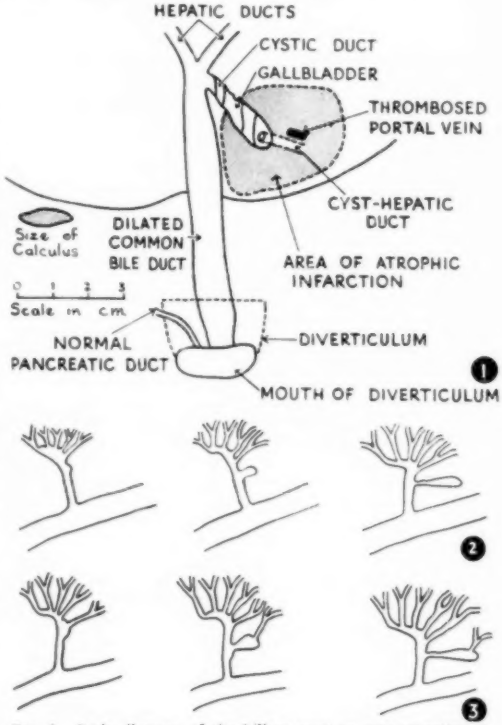

Fig. 1. Scale diagram of the biliary tract as seen at autopsy.
Fig. 2. Diagrammatic representation of the normal formation of the gall bladder.
Fig. 3. Diagrammatic representation of the method of formation of the cyst-hepatic duct at the time of formation of the gall bladder.

At no stage does there appear to be a direct communication between the liver and the gall bladder, and it has been suggested that the presence of cyst-hepatic ducts in man represents an atavistic reversion. It does appear, however, that there is one possible explanation for such a deviation from the normal. The usual method of development of the gall bladder is by the formation of a diverticulum from the common bile duct, just below the point where ramification of the hepatic ducts takes place (Fig. 2). If one postulates that such a diverticulum may arise slightly above the normal position, and such slight variations are often seen in the development of other systems in the human body, then it would appear that one, or more, hepatic ducts might be drawn out with the elongating gall bladder diverticulum and, depending on how far above the normal position the diverticulum originated, one could explain the occurrence of a cyst-hepatic duct at any

point from the fundus of the gall bladder to the junction of the cystic and common bile ducts (Fig. 3). This suggestion has quite possibly been advanced before, but as it was not found in the literature, it is put forward as a possible explanation since it is always more satisfactory to explain any abnormality on embryological rather than atavistic grounds.

DISCUSSION

In any possible case of congenital hypoplasia of the gall bladder it is very important to exclude a post-inflammatory shrinkage, which can be very marked. At first sight everything in this case might appear to favour the latter possibility—the history of an attack of jaundice two years before, jaundice at the time of death, a facetted calculus in the gall bladder and adhesions in the porta hepatis. The microscopic findings in the gall bladder wall are quite in keeping with the history, the other findings and the presence of a calculus in the gall bladder, but two points are in favour of a congenital origin. The first is the way in which the gall bladder is buried in the liver with no gall bladder bed distal to it; the other the presence of the cyst-hepatic duct. When dissection of the specimen was complete it was felt that this last feature outweighed all other considerations. The fact that it lay right at the tip of the gall bladder was felt to exclude the possibility of this being wholly, or even largely, post-inflammatory.

It is interesting to speculate on what would have been the patient's fate had she been admitted for investigation during the first attack of jaundice. In the first place it is unlikely that the gall bladder would have been seen on cholecystography, due both to its small size and to the presence of a large calculus in its lumen. If operation had been decided upon, it would have been technically very difficult, due to the position of the gall bladder and to the degree to which it was buried in the right lobe of the liver. Even had it been removed successfully, the cyst-hepatic duct must inevitably have been cut and ligation extremely difficult, if not impossible. Troublesome post-operative oozing of bile from the gall bladder bed is believed in some cases to be due to the presence of small cyst-hepatic ducts.

The dilatation of the common bile duct found is assumed to have been caused by a calculus which later was passed. The fact that the calculus in the gall bladder was facetted indicates that there must have been others present, and no other cause of obstruction to the biliary system was found. The diverticulum in the duodenum appears to have been congenital in type. The situation is typical, as is the histological appearance—a layer of mucous membrane separated from the pancreas only by a thin layer of loose connective tissue (Cole, 1920). There was no evidence of inflammatory change.

The red infarct in the liver was interpreted as being due to thrombosis of the portal vein which was in turn caused by a cholangitis of the closely related cyst-hepatic duct, presumably at the time of the previous attack of jaundice, though there is no proof of this.

SUMMARY

1. The case is presented of a Cape Coloured female of 74 with a true congenital hypoplasia of the gall bladder, biliary calculi, a cyst-hepatic duct and a duodenal diverticulum.

The cause of death was renal suppuration and subsequent spread of the infection, in a diabetic subject.

2. The scanty literature on hypoplasia of the gall bladder is reviewed.

3. The embryology of the biliary tract is presented briefly and a suggestion put forward to account for the origin of accessory hepatic ducts on the grounds of developmental anomaly rather than atavistic reversion.

I have to thank Dr. Helen Brown of the Department of Medicine, University of Cape Town, for access to the case records, Dr. J. A. Keen of the Department of Anatomy and Dr. J. H. Louw of the Department of Surgery for helpful criticism.

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ABSTRACT

Modern Treatment of Syphilis. Thomas, E. W. (1951): Bull. New York Acad. Med., **27**, 175.

Thomas believes that the treatment of syphilis is now comparatively simple. Even the great majority of late cases need no more than 15 days. He does not advise single large injections for the treatment of early syphilis though at Bellevue Hospital 85% of cases of primary syphilis responded well to a single injection of 2,400,000 units of Procaine Penicillin with oil and aluminium monostearate (PAM).

For early syphilis he advises an initial dose of 1,200,000 units of PAM followed by 5 daily injections of 600,000 units.

Late neurosyphilis can apparently be successfully arrested in over 90% of cases by 15 daily injections of 600,000 units of PAM.

For other types of late syphilis 6,000,000 units of PAM are given in 10 to 15 days, either 600,000 units daily or 1,200,000 units on alternate days.

For relapses he recommends re-treatment with double the initial dose.

Thomas does not favour treatment with the heavy metals but would rather try Aureomycin in doses of 4 gm. a day for 10 to 14 days. He admits that treatment with Aureomycin is still experimental. He found very few cases where Penicillin treatment had to be terminated on account of sensitivity to the drug.

Over 1,000 cases of active neurosyphilis as proved by spinal fluid examinations received Penicillin therapy or fever. He found abundant evidence that fever therapy was far superior to metal therapy in all forms of neurosyphilis, and now finds Penicillin just as effective as malaria. Permanently damaged nerve tissue naturally does not respond to any treatment. He agrees with Dattner that increased protein and cell counts indicate active neurosyphilis. It is still not possible to obtain seronegativity in at least 70% of patients for 10 years after the onset of late syphilis. Neurosyphilis rarely if ever relapses more than 2 years after the spinal fluid examination shows negativity.

He favours quantitative blood tests, but points out that there is no relationship between the amount of reagin found in the blood and the severity of the disease. Active syphilis can be found with low blood titres. Sharp rises in previously low titres are an indication for re-treatment, though a single rise does not prove relapse.

South African Medical Journal
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EDITORIAL

The Editor, the Medical Secretary and the Staff of the Head Office extend to all members of the Association the compliments of the season and best wishes for the new year.

ALCOHOLISM: A PUBLIC HEALTH PROBLEM

A recent report[1] of the establishment in Pretoria of a Central Organizing Committee for Conferences on Alcoholism under the patronage of Dr. Karl Bremer, M.P., Minister of Health and Social Welfare, emphasizes the increasing recognition of alcoholism as more than a clinical problem. The South African Central Committee will have members representing commerce, industry, the church, Government departments, hotel associations, the building trade and the international movement, Alcoholics Anonymous. This progressive step will undoubtedly receive wide support, as evidenced by the recent successful meeting arranged by the Organizing Committee in Pretoria from 20-21 November 1951.

That alcoholism is a public health problem, rather than a clinical or social disorder subject to reform by legal measures, is a view that is steadily gaining acceptance. When the Health Committee of the League of Nations tried in 1928 to deal with alcoholism as part of a world-wide health problem, its efforts were hampered by lack of sufficient understanding, on the part of the general public, of the medical and public health aspects of alcoholism. In the past 10 or 12 years, however, alcoholism has been the object of scientific research and noteworthy progress has been made in treating it as a disease affecting the mental and physical health of a considerable portion of the adult population of many countries. This progress enabled the Sub-Committee on Alcoholism of the WHO Expert Committee on Mental Health, when it met in December 1950, to base its discussions on objective data and to recommend to WHO and to national health administrations practical measures for preventing and treating alcoholism. The report of this Sub-Committee's discussions and recommendations has recently been released by WHO and is available as No. 42 in the *World Health Organization: Technical Report Series.*

1. *The Cape Argus*, 15 October 1951.

VAN DIE REDAKSIE

Die Redakteur, die Mediese Sekretaris en die personeel van die Hoofkantoor wens al die lede van die Vereniging 'n gelukkige Kersfees toe en alles van die beste vir die Nuwe Jaar.

ALKOHOLISME: 'N VRAAGSTUK VIR PUBLIEKE GESONDHEID

In 'n onlangse rapport[1] oor die aanstelling van 'n Organiserende Sentrale Komitee om Konferensies oor Alkoholisme te hou, onder voorsitterskap van dr. Karl Bremer, L.V., die Minister vir Gesondheid en Maatskaplike Welsyn, te Pretoria, word dit al hoe meer erken dat dranksug iets meer is as net 'n kliniese probleem. Die Suid-Afrikaanse Sentrale Komitee sal lede bevat verteenwoordigende die handelswese, industrie, die kerk, goewermentsdepartemente, hotelverenigings, die bougenootskap en die internasionale beweging, die *Alcoholics Anonymous.* Hierdie voorwaartse stap sal sekerlik wye ondersteuning geniet, soos op 20-21 November 1951 deur die suksesvolle vergadering, wat onlangs belê is te Pretoria deur die Organiserende Komitee, bewys is.

Dat verslaafdheid aan drank 'n probleem vir die openbare gesondheid is, liewers as 'n kliniese of 'n maatskaplike stoornis, wat reggemaak kan word deur wetgewing, is die beskouing wat al meer veld wen. Toe die Gesondheidskomitee van die Volkebond in 1928 probeer het om alkoholisme as deel van 'n wêreldwye gesondheidsvraagstuk te behandel, was hierdie pogings gehinder deur gebrek aan genoegsame begrip, van die kant van die gewone publiek, van die mediese en maatskaplike gesondheidsaspekte daarvan. Gedurende die laaste 10 of 12 jaar egter het alkoholisme die onderwerp geword vir wetenskaplike navorsing en is daar merkwaardige vooruitgang gemaak deur dit te behandel as 'n kwaal, wat die verstandelike en liggaamlike welsyn van 'n aansienlike deel van die volwasse bevolking in baie lande beïnvloed. Hierdie vooruitgang het die Sub-komitee oor Alkoholisme, van die WGO se Komitee van Deskundiges oor Geestesgesondheid, toe dit in Desember 1950 byeen gekom het, in staat gestel om sy besprekings te grond op objektiewe gegewens en om aan te beveel by die WGO en die nasionale gesondheidsadministrasies, praktiese stappe vir die voorkoming en behandeling van alkoholisme. Die verslag van hierdie Sub-komitee se besprekings en aanbevelings is onlangs vrygestel deur die WGO en is verkrygbaar as No. 42 in die *World Health Organization: Technical Report Series.*

1. *The Cape Argus*, 15 Oktober 1951.

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It is clear that research is still needed to determine whether psychological or physio-pathological factors are the basis of addictive drinking. Although recent studies have indicated that the addictive phase of alcoholism may have a physical basis of a constitutional nature, psychotherapy is indispensable in its treatment, since the excessive symptomatic drinking (which is a pre-requisite for addictive drinking) is psychogenic or sociogenic, or both. Psychotherapy will, in most cases, need to be supplemented by the type of aid which a social worker can supply.

Certain drugs may prove to be a valuable adjunct to psychological and social therapy. Chief among these is tetraethylthiuramdisulphide, which causes a severe somatic reaction if the patient drinks alcoholic beverages when he is on a maintenance dose of the drug. This preparation should be available only to the medical profession and should preferably be employed only on cases in which continued supervision is possible. In more advanced cases of addictive drinking, other techniques such as the various forms of 'aversion' therapy—based on the use of apomorphine or emetine—may be more valuable. Even hormones such as ACTH or Cortisone have been reported to be effective in treating delirium tremens, acute alcoholic intoxication, and Korsakoff psychosis.

Dit is duidelik dat daar nog behoefte bestaan aan navorsing om vas te stel of sielkundige of fisio-patologiese faktore ten grondslag lê aan drankverslaafdheid. Hoewel resente navorsing aangedui het dat die fase van verslaafde drankgebruik 'n fisieke grondslag van konstitusionele aard mag hê, tog is psigoterapie onmisbaar vir die behandeling daarvan, want die buitensporige simptomatiese drankmisbruik (wat 'n voorwaarde is vir verslaafde drankgebruik) is van 'n sielkundige of maatskaplike aard, of beide. Psigoterapie sal in die gros van gevalle ook aangevul moet word deur hulp van die soort, wat 'n maatskaplike werker kan bied.

Sekere medisynes mag as waardevolle, newe hulpmiddels, naas sielkundige en sosiale behandeling dien. Die vernaamste een is tetra-etiel-tiuram-disulfied. Dit veroorsaak 'n geweldige liggaamlike reaksie wanneer die pasiënt alkoholiese dranke neem onderwyl hy 'n onderhoudsdosis aan die gebruik is. Hierdie middel behoort slegs in hande van die mediese beroep te wees en moet liefs gebruik word, slegs vir gevalle wat onder volgehoude toesig kan staan. By meer gevorderde gevalle wat aan drank verslaaf is, mag ander metodes waardevoller wees, soos bv. die verskillende vorms van ‚weersinverwekkende' behandeling wat gebaseer is op die aanwending van apomorfien of emetien. Verslae het selfs al verskyn dat hormone, soos ACTH of Kortisoon, doeltreffend is by die behandeling van dronkenskapsdelirium, akute vergiftiging deur alkohol en die Korsakoffse sielsiekte.

AGRANULOCYTOSIS DUE TO ANTIHISTAMINE DRUGS

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Agranulocytosis due to antihistamine drugs is rare, only seven cases having been recorded (Blanton and Owens, 1947; Clement and Godlewski, 1945; Cahan *et al.*, 1949; Drake, 1950; Hilker, 1950; Martland and Guck, 1950) despite the extensive use of these drugs in recent years. It is thus thought worth while recording a case of agranulocytosis due to one of this group of drugs.

CASE REPORT

A 66-year-old Jewish widow was first seen on 24 February 1950 for severe coryza. Her previous history revealed that in August 1949 a cholecystectomy was performed for gall stones, with an uneventful recovery. Three weeks before the operation she had an attack of acute bronchitis, and was treated for five days with a compound sulphonamide, Sulphatriad, containing Sulphadiazine 0.185 gm., Sulphamerazine 0.13 gm. and sulphathiazole 0.185 gm. per tablet. One week before operation the blood urea was 23 mg. per 100 c.c., the prothrombin index was 95% and the haemoglobin 14.1 gm. per 100 c.c. of blood.

Her present complaints were severe nasal discharge and an irritating non-productive cough. There were no abnormal physical signs, apart from pyrexia of 99.4° F. Therapy consisted of a 'cough' syrup containing equal parts of 'Anodyne Pine Expectorant' Parke Davis (a mixture of various bark and root extracts with 3/16 grain of morphine acetate to the ounce) and 'Syrup Cocillana Co. B.P.C. 1949' to be taken in 1 drachm doses every four hours, and two tablets of Empirin Compound (Aspirin 3½ gr., Phenacetin 2½ gr. and Caffeine ½ gr. per tablet) twice daily, as well as 25 mg. of Phenendamine (Thephorin) thrice daily for four days. The condition abated rapidly, and within three days, she was well except for a slight cough. When seen again on 10 March, she stated that for a few days she had experienced weakness, a feeling of fullness above the eyes, cough productive of yellowish sputum, a tearing pain under the sternum on coughing, and pain deep to the xiphisternum on swallowing foods. She also complained that food did not go down easily, and that she had a continual feeling of pressure under the lower third of the sternum.

The temperature was 100.4° F., the pulse rate 120 per minute, and the blood pressure 130/80 mm. Hg. There were a few scattered medium crepitations at both lung bases. The area of stomach resonance appeared to be higher than usual. The diagnosis made was that of an acute upper respiratory infection involving the sinuses, trachea and bronchi. A liquid mixture of sulphonamide containing Sulphadiazine 5 gm. and Sulphathiazole 5 gm. to the 100 c.c., in a base containing sodium citrate

(Aldiazol), was prescribed. The initial dose contained 2 gm. of sulphonamide, and this was followed by 1 gm. every four hours. As the mixture caused vomiting, she discontinued the medication after having taken less than 12 gm. of sulphonamide. Ephedrine 1% in normal saline as nose drops, and Syrup Codein Phosphate B.P.C., one drachm 4-hourly were also prescribed. On the following day, the feeling of fullness above the eyes had disappeared, and the temperature had fallen to 99° F., but the other symptoms persisted. For the next few days the temperature continued between 99° F. and 100° F., and as the other symptoms including cough, and tachycardia became worse, she was admitted to a private hospital on 17 March 1950 for further study.

X-ray examination of the chest showed the pulmonary pattern to be rather more prominent than average, this being interpreted as being due to 'catarrhal' changes, and there was slight widening and calcification in the aortic arch. An X-ray examination of the oesophagus and upper gastro-intestinal tract revealed that she swallowed considerable quantities of air during the examination, but the oesophagus and upper gastro-intestinal tract appeared normal.

On examination, the physical signs were unchanged, the temperature was 100.4° F., the pulse rate 100 per minute and her weight was 135 lb. There were no ulcerations in the mouth. Agglutination tests for the typhoid group, brucellosis and rickettsial infections were negative. Cold agglutinins were not present. Malarial parasites were not present in thick smears, and the urine was normal on routine testing. An electrocardiogram was normal in all respects. She was given 0.5 gm. Aureomycin 4-hourly by mouth, but this was stopped after two days because of nausea. On 18 March she was started on 500,000 units aqueous procaine penicillin G, 12-hourly, by intramuscular injection. The report of a blood count taken on 17 March (received on 20 March) showed a haemoglobin of 12 gm.%; C.I., 1.05, erythrocytes 3,700,000 per c.mm., and leucocytes 850 per c.mm. In the differential count only 40 leucocytes were noted, of which one was a neutrophil. A myelogram of the sternal marrow on 20 March (Dr. J. Gluckman) showed blast cells 3%, premyelocytes 1%, myelocytes 6%, metamyelocytes 6%, stab cells 3%, polymorphonuclears 0%, lymphocytes 26%, monocytes 5%, erythroblasts 1%, proerythroblasts 4%, early normoblasts 5%, intermediate normoblasts 20%, late normoblasts 20%. This was interpreted as showing 'a severe depression of the granular series with defective maturation.' There was no disturbance of the erythropoietic mechanism, with a relative increase in that series. This, coupled with the absence of any 'leukaemic hiatus' and a normal thrombocytic system, pointed to a 'toxic agranulocytosis', rather than to a 'subleukaemic leukaemic state.'

These facts indicated a malignant neutropenia, and the treatment for agranulocytosis was accordingly instituted. Therapy consisted of ½ gm. Streptomycin and 500,000 units of crystalline Penicillin 6-hourly, and 10 c.c. of pentnucleotide intramuscularly 6-hourly. Twenty micrograms of vitamin B_{12} daily by injection, and 30 mg. of folic acid daily by mouth, were also given. Two blood transfusions of 500 c.c. of citrated blood were given on 20 March and 23 March. The clinical course was stormy. Until 20 March the temperature varied between 99° F. and 103° F. On 20 March slight ulceration round her only tooth—a right lower molar—was seen, but no other ulceration was present. Cough and painful dysphagia were troublesome until 27 March, when improvement started and permitted a reduction in the frequency of the pentnucleotide and antibiotic injections to 8-hourly injections. A pyrexia of 103° F. to 104° F. continued until 26 March, when it started to come down gradually, and by 29 March it returned to normal. On 23 March she developed sordes on the lips, and on 27 March a solitary pin-point ulcer appeared at the junction of the hard and soft palates. At times she was delirious. Profuse night sweats commenced on 26 March and recurred nightly until the temperature settled on 29 March. By 30 March, the sordes had disappeared, the palatal ulcer was healed and the patient was ambulatory. On 31 March the pentnucleotide was reduced to 10 c.c. twice daily because of sweating and vomiting following the injections, but the antibiotics were continued 8-hourly until 3 April. On 4 April, pentnucleotide was reduced to 10 c.c. daily and was finally discontinued on 9 April. By 5 April, ulceration round the tooth had disappeared, and the patient felt much stronger.

Further clinical progress was uneventful until her discharge on 22 April, when she weighed 131 lb., compared to 135 lb. on admission. The Penicillin and Streptomycin were reduced to 12-hourly injections on 3 April, and were reduced in dose to 250,000 units penicillin and 0.25 gm. Streptomycin twice daily on 10 April. On 12 April, 2 c.c. crude liver extract and 1 c.c. vitamin B compound were given daily by injection, as well as 2 capsules of Campobiol (vitamin B Compound and 100 mg. ferrous sulphate per capsule) orally thrice daily. On 14 April, the Streptomycin and crystalline Penicillin were stopped, and replaced by one daily injection of 600,000 units of aqueous Procaine Penicillin G. On 19 April all injections were stopped, but she took oral Campobiol and folic acid until her discharge on 22 April.

The clinical improvement was paralleled by the haematological picture, and from 27 March, there was a progressive increase in the percentage and absolute numbers of neutrophils, although the total leucocyte count remained below 1,500 per c.mm. for a further week. (Table 1 and Fig. 3.) From then onwards, the total leucocyte count and absolute number of neutrophils continued to increase. On 3 May the leucocyte count was 3,300 per c.mm., but the sternal bone marrow reflected a dramatic return to normal haematopoiesis, although the marrow was still hypoplastic. The myelogram showed a total nucleated count of 18,000 with blast cells 1%, premyelocytes 2%, myelocytes 3%, metamyelocytes 6%, metamyelocyte eosinophils 2%, stab cells 10%, polymorphonuclears 32%, polymorphonuclear eosinophils 4%, lymphocytes 18%, monocytes 2%, erythroblasts 1%, proerythroblasts 1%, normoblasts—early 4%, intermediate 6%, late 7%—plasma cells 1%.

On 4 June she developed an upper respiratory infection with a temperature of 100.4° F. Her leucocyte count at this time was 6,700 per c.mm. with 76% neutrophils, indicating a return to normality. The following day her temperature was normal, and she has remained well.

TABLE I

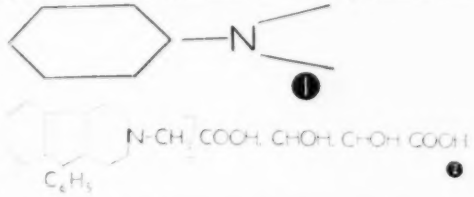
Date	Hb %	Red Blood Cells (per c.mm.)	C.I.	White Blood Cells (per c.mm.)	N	M	L	E	B
17 March	12	3,700,000	1·05	850	2	–	—	–	–
20 March	11·7	4,420,000	0·88	600	6	12	82	–	–
21 March	12·0	4,120,000	0·95	800	6	2	92	–	–
22 March	11·3	3,780,000	0·98	800	—	–	—	–	–
23 March	11·1	3,740,000	0·96	500	4	8	88	–	–
24 March	12·5	4,280,000	0·95	600	14	6	80	–	–
25 March	12·7	4,240,000	0·97	500	21	9	70	–	–
26 March	12·5	4,500,000	0·95	1,000	27	1	70	2	–
27 March	12·5	4,120,000	0·99	900	37	6	57	–	–
28 March	11·6	4,360,000	—	600	40	2	56	2	–
29 March	13·3	4,600,000	0·95	1,300	32	10	56	1	1
30 March	13·1	4,720,000	0·90	1,200	52	9	39	–	–
1 April	13·7	4,680,000	0·96	1,100	53	7	40	–	–
3 April	13·2	4,680,000	0·92	1,100	48	4	47	1	–
5 April	13·1	4,360,000	0·98	900	39	2	53	4	2
6 April	12·7	4,020,000	1·01	1,500	52	6	38	3	1
9 April	13·0	4,510,000	1·00	2,600	42	2	53	2	1
11 April	13·0	4,020,000	1·03	1,900	49	10	36	4	1
13 April	12·4	3,840,000	1·05	2,500	51	13	34	2	–
15 April	12·4	4,240,000	0·95	2,000	67	6	25	2	–
18 April	13·3	4,360,000	0·99	4,200	58	6	36	–	–
22 April	11·7	4,170,000	0·97	3,500	52	11	33	4	–
3 May	10·6	4,000,000	0·91	3,300	53	3	39	5	–

DISCUSSION

Although it is impossible to say with certainty that the antihistaminic was responsible for the agranulocytosis, it appears to be the likely cause. It cannot be denied that the course of sulphonamides in August 1949 may have 'sensitized' her, and that the sulphonamide mixture given in March 1950 may have been the etiological factor, but against this is the fact that the symptoms, including dysphagia, were already present when the sulphonamides were started. The phenacetin in the Empirin compound can be dismissed as a cause because, after her recovery from the agranulocytosis, she had repeated doses of this drug for the upper respiratory infection in June 1950, with no adverse effect on the neutrophil response. The only proof would be a therapeutic trial with the Phenendamine, but in view of the serious nature of the disease this procedure must be deemed unjustifiable.

Seven cases of agranulocytosis due to antihistaminic drugs have been reported in the literature. In the reported cases the period of administration has been from three to eight weeks, and the present case would appear to be unusual in that only 300 mg. of Phenendamine were taken over five days. Blanton and Owens (1947) reported the case of a widow aged 73, who was given 10.75 gm. of Tripellanamine (Pyribenzamine) over a period of eight weeks for urticaria. Her white cell count went down to 1,300 per c.mm. with 3% neutrophils. After withdrawal of the drug and the intramuscular use of Penicillin the temperature dropped to normal in 12 hours, and the blood count returned to normal in 12 days. Cahan *et al.* (1949) described a case of agranulocytosis due to Pyribenzamine in a 39-year-old woman, with a contact dermatitis. She took 4.0 gm. of Pyribenzamine over a period of 5½ weeks. Her white cell count dropped to 1,700 with 2% neutrophils. She made a rapid recovery on treatment with intramuscular Penicillin. These writers also mention another case of a patient who took approximately 400 mg. of Pyribenzamine for a month before the onset of agranulocytosis. The white cell count declined to 1,100 and the neutrophils disappeared completely. She made an uneventful recovery. Drake (1950) reported a case of agranulocytosis in a man aged 81 who suffered from an allergic condition of the nasal mucosa, and took approximately 7.4 gm. of Methaphenilene (Diatrin) over a period of 6½ weeks. His white cell count went down to 1,000 with complete disappearance of the neutrophils. He made a good recovery on treatment with Penicillin and Dihidrostreptomycin. Clement and Godlewski (1945) reported the first case of agranulocytosis due to an antihistamine drug in a girl of 13½ years, with asthma, after she had received three weeks of treatment with a synthetic antihistamine 2339 RP (N-Benzyl-N-phenyl-N, N-dimethyl-ethylenediamene-chydrochloride). Two further cases have been reported both due to Pyribenzamine (Hilker, 1950; Martland and Guck, 1950). In one case 50 mg. was taken three times daily for eight weeks by a 39-year-old nurse, and in the other, eighty-five 50 mg. tablets were taken over a five-week period by a female aged 60 years. Neither case was fatal. Thus, of the reported cases, the drug used was Pyribenzamine in five, Methaphenilene (Diatrin) in one, and antihistamine agent 2339 RP in the remaining case. The present case would appear to be the first due to Phenendamine. Fortunately all eight cases have recovered.

In the production of agranulocytosis Kracke (1934) has stressed the importance of the benzene ring in combination with nitrogen or the amino (NH_2) group, making substituted primary amines. Many of the antihistaminic agents, such as Pyribenzamine and Methaphenilene, contain this so-called 'benzamine linkage' (Fig. 1), as do some of the sulphonamides, but others such as Diphenhydramine (Benadryl) have no such linkage, and have never been known to cause agranulocytosis. Other antihistaminics such as Mepyramine maleate (Anthisan) contain a 'benzamine linkage' in their structure, but despite extensive use, have not as yet been reported to cause agranulocytosis. Both Tripellenamine (Pyribenzamine) and Methaphenilene (Diatrin) are ethylenediamines, but Tripellenamine contains a pyridine ring structure, as do Anthisan, Histadyl and Thenylene. Methaphenilene (Diatrin) differs from the above group by substitution of a phenyl ring for the pyridine structure (Loew, 1950). Phenendamine (Thephorin) is not closely chemically related to any of the other antihistamine agents (Loew, 1950), and study of this structure indicates that it does not contain a benzamine linkage (Fig. 2). It has the chemical designation of 2-methyl-9 phenyl-tetra-hydro-pyridinene hydrogen tartrate. This structure contains two



benzene rings, as does the agent 2339 RP used by Clement and Godlewski (1945) and Drake (1950) points out the possibility that such a chemical structure increases the probability of granulocytopenic effect. It is thus obvious, owing to the widely different chemical characteristics of the antihistamine group of agents, and the fact that some with a 'benzamine linkage' have produced agranulocytosis while others have not as yet done so, that only future experience will tell which are capable of causing granulocytopenic effects.

There are other interesting features in the present case. Damashek (1944) stated that 'a patient (with agranulocytosis) over 60 years of age has hardly any chance'. Furthermore, the lower the total leucocyte count the worse the outlook, recovery being exceptional with counts under 1,000 per c.mm. He considered a complete lack of granulocytes to be of no great prognostic significance. In the present case, despite the patient's being over 60 years and the count going as low as 500 on two occasions, she made a good recovery. This must be attributed to modern antibiotic treatment. The type of marrow found in this case with the presence of granulocyte precursors indicates a better outlook, than in those cases in which only lymphocytes are found (Damashek, 1944).

A further feature of interest in this case is the paucity of ulcerations in the mouth, and the presence of painful dysphagia as the main symptom.

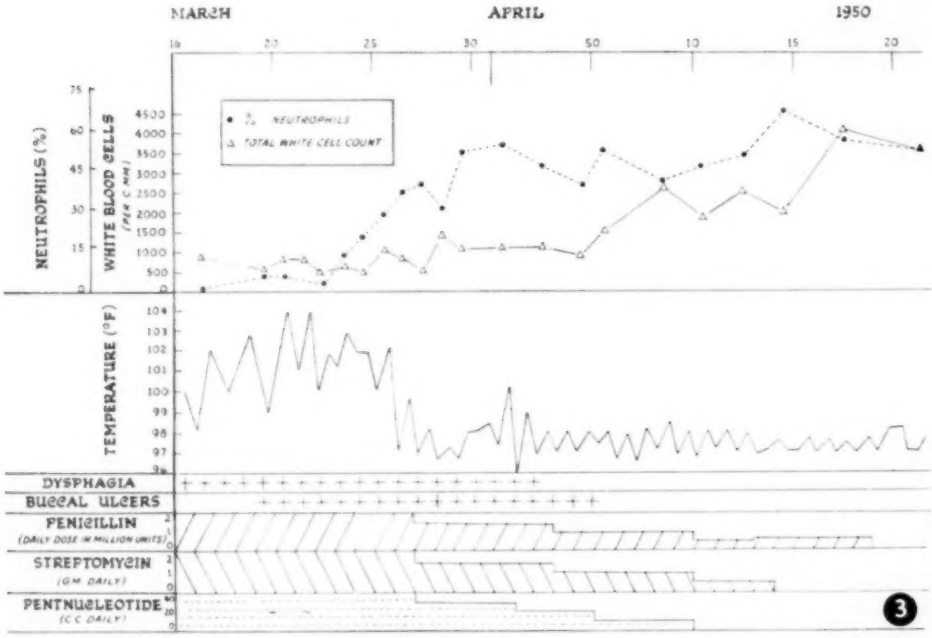
SUMMARY

1. A case of agranulocytosis has been recorded.

2. The possibility that the condition was due to an antihistamine drug has been discussed.

3. Seven other cases reported in the literature are reviewed, making a total of eight cases to date in which antihistamine drugs could be inculpated as the cause of agranulocytosis.

The writer wishes to acknowledge the invaluable help and advice of Dr. M. M. Suzman who saw this case in consultation with him and gave many valuable suggestions in connexion with this paper.



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A CASE OF METHYL BROMIDE POISONING

SIMULATING RUPTURE OF AN INTRACRANIAL ANEURYSM.*

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Of several comprehensive studies dealing with methyl bromide (CH_3Br) poisoning, those by Von Oettingen [1] and Johnstone [2] excel. It is generally agreed that effects in the living body and in experimental animals depend on the site of entrance this being, in the case of methyl bromide, usually the air passages. Damage to the skin caused by exposure, e.g. in manufacturing plants, to vapours of this volatile substance is well known; this, however, plays a minor role.

It is convenient to distinguish between the local damage to the organs of the respiratory tract, especially the lungs, and the effects which are brought about after the absorption of the poison into the body.

Local damage to the lungs is mainly characterized by pulmonary oedema which eventually may prove fatal. The symptoms and signs that make their appearance after absorption of the gas indicate that the nervous system is mainly involved. Consequently it is the neurological findings upon which the diagnosis has to be based (Von Oettingen [1]). A somewhat confusing variety of symptoms of central nervous system origin has been described, such as epileptiform convulsions, myoclonic twitchings, tremor, adiadokokinesis. French authors (Michaux *et al.*[4]) mentioned cerebral oedema in one fatal case.

It is an interesting and rather complex group of involuntary movements of the myoclonic or choreic type to which the author of this paper wishes to draw attention; moreover, the following case, observed in the Neurosurgical University Clinic in Zürich, exhibited signs of acute meningeal haemorrhage and consequently gave rise to differential diagnostic considerations that seem to be of some practical value. Finally, the case described here is probably the first instance of methyl bromide poisoning in man, during the course of which EEG records have been taken. The above-mentioned pattern of involuntary movements has also been studied by means of motion pictures.†

Case History. G. J., aged 32, automobile dealer, suffered an injury to his head (occipital region) in 1943 and subsequently he complained for about six months of headaches. One day he passed into a convulsive state which was followed by unconsciousness; this was, however, a single attack and did not recur.

Six months before admission to hospital, he had started handling automobile fire extinguishers which contained a mixture of methyl-ethyl bromide. He emptied and refilled the containers in a cellar without proper ventilation. He had never complained of any troubles previously. On the afternoon of 27 February 1949, when doing his usual work, he became dizzy, developed headaches, and left the room for his flat. There, he developed nausea, vomited and lay down. In the evening, his wife summoned the house doctor. By this time, the patient was confused and disorientated, his skin was pale and clammy, he breathed heavily and was scarcely responsive. His pupils showed a slow reaction to light, he was unable to open his mouth fully, to extrude his tongue or to whistle. There was a doubtful motor paresis and loss of sensation in the right arm. The reflexes were hyperactive and equal and ankle clonus was noted. Both plantar responses were flexor. Shortly afterwards, the patient had generalized convulsions which (according to his wife) started with a sudden turning up of the eyes, pulling up of the arms and groaning. Twitchings of the legs and trunk followed. Some frothy saliva was extruded from between the lips, but no incontinence of urine was noticed. Subsequently a series of similar paroxysms occurred and the patient was sent as an 'emergency case' to the Canton Hospital. On admission he was unresponsive and agitated, each convulsion was followed by complete unconsciousness, and he did not react to painful stimuli. There was evidence of acute pulmonary oedema which cleared up within a few hours.

Neurological examination revealed reflexes equally hyperactive on either side, and sometimes ankle clonus was present. The paroxysms were of generalizd tonic and clonic character, the patient did not bite his tongue, and the plantar responses were flexor immediately following the fits. Between the seizures there was a striking change in his behaviour, this latter being bizarre and clownish. A lumbar puncture was done and the pressure found to be 17 cm. H_2O; the fluid was mixed with fresh blood. The next day, the attacks recurred and the plantar response was now extensor on the right. The following day the seizures ceased and gradually the patient regained consciousness and began to take fluids; the right plantar response was still extensor. It was now stated that the patient exhibited an almost complete amnesia for events of the past two days and his reactions and responses were slow. The right plantar reflex became flexor. *A brain aneurysm with subarachnoid haemorrhage* was suspected and the patient was transferred to the Neurosurgical Clinic.

On admission there, on 2 August, he complained of feeling dizzy but was not nauseated and did not have headache. After reading for a few minutes he tired and his vision became blurred; there was, however, no double vision. There was increased fatiguability and his power of thinking and of remembering facts was depressed. He complained especially of a sort of clumsiness as well as of trembling and involuntary twitchings of his left hand and fingers. There was some sensory loss, and

* From the Neurosurgical University Clinic in Zürich, Switzerland. Director Prof. Dr. H. Krayenbühl.
† The film is available and can be borrowed from the author of this paper.

occasionally he dropped objects from his left hand. He co-operated well. His temper was exhilarated and somewhat euphoric.

Physical examination revealed no stiffness of the neck; the fundi were within normal limits; the visual fields were normal on routine examination. The pupils were equal on both sides, with brisk reaction to light and on convergence. We found a slight divergent squint of the left eye but no nystagmus; the corneal reflexes were present. The left angle of the mouth slightly drooped and the left palpebral fissure was a little broader than the right. Speech was perhaps slightly slurred; but his wife said that this did not differ from his usual speech.

The most striking feature about the limbs was a type of more or less continuous involuntary movement of the left arm, hand and fingers which was more prominent in the distal parts of the limb. This almost ceased during rest and returned as soon as the patient started carrying out purposive movements, e.g., stretching out his arms and holding them in a horizontal position, grasping or picking up small objects, and so forth. Consequently the patient was unable to use his left hand and fingers appropriately. These spontaneous movements could not be inhibited or controlled by the patient and it seemed that emotional excitement exaggerated them. They were of a somewhat complex nature and consisted:

1. Of inco-ordinate twitchings similar to fasciculation of a single muscle of the forearm.

2. Of a choreic element which was especially pronounced in the hand and fingers. A proper athetotic component, however, seemed to be absent.

The right upper limb participated in these movements but only to a small degree; no such movements were to be found on the trunk and the legs. There was no striking difference in the muscle tone of the limbs. Furthermore, we found a distinct weakness of the left elbow and wrist (the patient being right-handed). Performance of rapid alternating movements such as pronation and supination of the forearm on the left was comparatively slower and clumsier. The rebound phenomenon was negative. In the finger-nose, and finger-nose-finger test, on the right, there was no past-pointing, but on the left, this movement was quite irregular and jerky. When touching the point of the nose, the left forefinger kept moving irregularly to and fro.

The tendon reflexes in the arms were hyperactive and equal on both sides. Hoffmann's snapping reflex and Trömner's finger phenomenon could be elicited on either side. In both hands there was a slight loss of the sensation of touch but that for pain was normal, the affected area not being sharply circumscribed. Joint vibration and two-point discrimination sense, were accurate and stereognosis was intact. The abdominal reflexes were equal. Low in the back, on flexing the thighs, the patient felt some pain due to a recent lumbar puncture.

Tone, movements and muscular power in the legs were undisturbed and the 'heel-knee-shin' test was faultless. Knee and ankle jerks were somewhat hyperactive and symmetrical. Muscle and nerve trunks were fairly tender on pressure. Gait was undisturbed and Romberg's sign was negative. The blood pressure was 110 mm. Hg systolic. Cerebral angiography was done on both sides and no aneurysm or vascular abnormality could be visualized.

On 8 August 1949 (the twelfth day after the onset of symptoms) the patient suffered another epileptic seizure which again was followed by complete unconsciousness. Convulsions, however, were not observed. Two days later air encephalography was done. The fluid was yellow and contained 22 mg. protein per 100 c.c. and 7 white cells per c.mm.; colloidal reaction of the cerebrospinal fluid was normal. The ventricles were found to be normal. EEG studies (taken on three different occasions) exhibited severe generalized dysrhythmia with bilateral 2 per second waves especially over the frontal regions (Fig. 1). After Luminal administration the involuntary movements greatly improved.

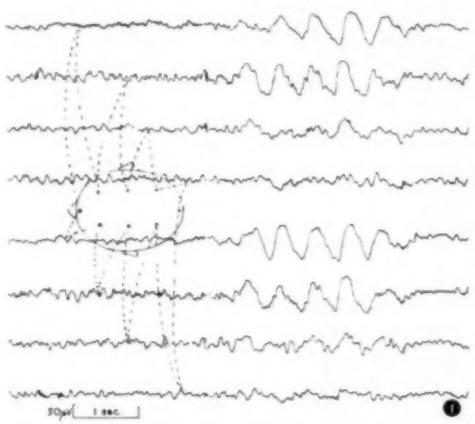

Fig. 1. EEG reading in a 32-year-old man. Subacute phase of intoxication by inhaling a mixture of methyl bromide containing gas.—Note outbursts of high two-second waves and theta-rhythm especially in the frontal and postcentral areas.

At a check-up examination on 1 February 1951, the patient complained that he stuttered when emotionally upset and consequently groped for words and he has become somewhat forgetful. There were no further fits. When handling small objects he was liable to lose control of his hands and fingers and start trembling; this gradually became worse when he tried to concentrate on his work. The neurological findings were essentially normal except for some unsteadiness and a delicate tremor and dysmetria of the left hand especially during the finger-nose-finger test.

On 12 June 1951, he reported that he still did not feel quite well. Though doing his professional work as a car-driver, he would frequently have trouble with his hand and fingers as they were trembling and jerky, and consequently were unfitted for delicate work. Moreover, he was somewhat irritable and emotionally unbalanced and, according to his wife, showed a slight change in character.

DISCUSSION

1. Reports on post-mortem findings in cases of methyl bromide poisoning record small haemorrhages and areas of softening in the brain. I found, however, but one instance where it was stated that the cerebrospinal fluid contained blood.[6] On the contrary, according to Johnstone, 'the spinal fluid has usually been found clear and of normal composition'; hence this writer concludes that the poisoning effect is due more to injury of the ganglionic cells and less to circulatory disturbances of the brain. French authors reported discrete oedema of the cerebral peduncles with vascular congestion. As yet, apparently not a single case of methyl bromide poisoning has been associated with such massive meningeal haemorrhage as to suggest rupture of an intracranial aneurysm. In this respect, at least, our observation appears to be singular, and the differential diagnosis may seem somewhat unjustified and even questionable. However, it had to be considered that, quite exceptionally, in this case there could be a combination of poisoning with rupture of an intracranial aneurysm. With the sequence of the case history in mind, however, I do not feel inclined to assume that in the case described there was an intracranial aneurysm present at all. The reasons are the following:

(a) The haemorrhage occurred in close relation to the acute damage and did not recur.

(b) There were no neurological symptoms at any time in favour of an aneurysm of the basal arteries. Any involvement of cranial nerves (oculomotor, trigeminal) so frequently associated with a basal aneurysm of the brain, was lacking.

(c) Cerebral angiography failed to show evidence of an intracranial vascular abnormality which, quite naturally, does not exclude aneurysm but makes it less probable. Thus there can be little doubt that we are dealing here with an exceptionally extensive bleeding, originating from the intracranial vessels, brought about by acute methyl bromide intoxication. On the other hand, the neurological symptoms observed can be more easily related to some sort of diffuse damage to the subcortical structures of the brain. Von Oettingen,[1, 7] emphasizes pathological changes in the lower section of the brain, the basal ganglia, cerebellum and the pyramidal tract.

2. The very weak narcotic action of methyl bromide[6] is fully confirmed by our observation. Actually there was no narcotic effect at all. Instead there was a peculiar depression and an alteration of the state of consciousness —his behaviour being described as 'bizarre and clownish' —somewhat comparable to epileptiform (psychomotor) equivalents. This state was transient and developed into true epileptic convulsions leaving the patient amnesic for a period of about two days.

3. EEG records taken during the subacute stage of the intoxication, exhibited severe bilateral functional disorder of the cortex. Peculiarly enough, there was but little evidence of cortical damage in the sensory and motor fields. Air studies revealed no gross defect in the brain parenchyma or distortion of the ventricles. We were not able to repeat the air encephalography.

4. Several authors[1, 8] describe a latent period before the onset of severe cerebral and other symptoms. I feel we too can generally confirm this phenomenon, since several hours elapsed before the patient was seized with generalized convulsions. It must be noted, however, that the patient's amnesia extended through that period, thus indicating—as one may assume—that even during that 'latency' there must have been developing some sort of cerebral depression.

5. Johnstone[3] regards tremor and apathy as late symptoms. He remarks: 'It must be regarded as highly questionable whether any pathologic change resulting from intoxication will be of permanent character. On the other hand, Von Oettingen,[7] summarizing reports up to 1946, states that certain neurological abnormalities such as nervousness, paresthesias, blurred vision and so forth may persist and incapacitate the individual for a long time. In accordance with this statement, the acute intoxication in our case resulted in long-standing symptoms which seem to indicate a more diffuse cerebral functional disorder (depression of memory, changes in behaviour and character) as well as noticeable damage to the sub-cortical centres (intention tremor and hyperkinetic phenomena).

SUMMARY

A case of acute poisoning by inhalation of a mixture of methyl bromide in a man of 32 years is described. This case has been followed up for about two years. Air encephalography, EEG records and cerebral arteriography were made in the subacute stage of the intoxication.

The main features, different to a certain extent from those related in the literature, were the following:

(a) *The acute phase* (viz., two days following the acute damage): There were initial transient pulmonary oedema, alteration of consciousness and behaviour, resembling an epileptic equivalent. After several hours there was a sudden onset of generalized convulsive attacks which recurred on the following two days, leaving the patient amnesic. Slight transitory, one-sided reflex and motor disturbances of the pyramidal tract (Babinski sign) were noted. In the cerebrospinal fluid, there was fresh blood indicating intracranial haemorrhage, suspicious of a brain aneurysm.

(b) *The subacute (intermediary) stage*: After a slow recovery and a single attack on the twelfth day, left-sided hyperkinetic phenomena (intention tremor, adiadokokinesis, involuntary movements of choreic and myoclonic type) developed in the distal parts.

(c) *The chronic course*. There was slow improvement with residual symptoms such as irritability, loss of memory, slight visual and speech disturbances, marked tremor and slight dysmetria in the left hand.

The mechanism of methyl bromide poisoning and the method of dealing with its prevention and treatment are discussed in the monographs by Von Oettingen[1] and Johnstone[2, 3]. This case did not reveal any remarkable features. It may be mentioned here that the administration of Luminal seemed to be useful in controlling the motor disturbances.

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ACUTE PORPHYRIA IN A BANTU MALE

A CLINICAL AND CHEMICAL REPORT

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Acute porphyria was first described by Ranking and Pardington in 1890,[1] and since that date numerous cases of this uncommon metabolic disturbance have been described in many parts of the world.

Waldenström[2] by 1937 had found 89 cases in the literature, and to these he added his own 103 Swedish cases. With the exception of five from the United States of America all these had occurred in Europe. Since then many accounts of single and small groups of cases have been recorded from many sources. South African cases have been reported by Kooy,[3] Linder[4] and Barnes.[5, 5a]

Porphyria in man occurs in three syndromes:

1. The first is the rare congenital porphyria characterized by photosensitivity of the skin, pigmentation of the bones and teeth and passage in the urine of uroporphyrin type I. The symptoms are recognized in infancy.

2. The second is the more common acute porphyria which is manifested by susceptible persons in the form of recurrent attacks often apparently precipitated by periods of stress. Clinical symptoms, which vary considerably from case to case, include acute abdominal or neuritic pain; constipation; vomiting; muscular wasting, weakness or paralysis and variable psychotic manifestations. Not all of these need be present. The urine during attacks contains porphobilinogen and porphyrin, usually in considerable amount though the latter may only become evident on standing. The porphyrin was regarded as being uroporphyrin III, but more recent investigations indicate that several of these pigments are present.

3. The third type, porphyria cutanea tarda, is characterized by a dermatitis, frequently bullous in type, which becomes manifest in adult life. The pink or brownish urine contains porphyrin but no porphobilinogen. Neurological and psychotic symptoms are not present.

Some cases have been reported which appear to be a combination of the last two forms, e.g. Gray *et al.*[6]

The case to be described falls into the second category and, as the clinical picture varies, some additional facts will be described. The classical symptoms have already been mentioned, but it must be stressed that they do not necessarily occur simultaneously, and nervous features in particular may never be present. The urine, which is usually port wine in colour, may be normal when passed and darken to almost blackness on exposure to sunlight.[7] The abdominal pain, often accompanied by visible peristalsis, may, in the absence of neurological symptoms and discoloration of the urine, be attributed to an abdominal catastrophe. Waldenström mentions 29 cases of acute porphyria in which a laparotomy was performed. Constipation is more usual, but diarrhoea may be present in some cases.

The nervous symptoms usually do not fit into any definite syndrome. The two common manifestations are either a condition resembling Landry's paralysis, or a bizarre type of polyneuritis. These may be accompanied by varying psychological disorders, particularly confusion, hallucinations or occasionally coma. At times the psychological manifestations overshadow the physical accompaniments. Hypertension is a common finding.

The mortality rate is about 50%; but where the nervous system is involved it is thought to be even higher.[8]

CASE REPORT

On June 17 1950 a Native male, aged 24 years, was admitted to the King Edward VIII Hospital, Durban, complaining of abdominal pain and weakness of all his limbs, especially his arms.

History. The man stated that three weeks before admission he had developed severe abdominal pain. The pain was intermittent and moved all over the abdomen, being associated with vomiting and hiccough. Neither constipation nor diarrhoea had been present. Two weeks after the abdominal pain had started he developed pain in the legs, which was followed shortly by weakness and difficulty in walking. At this stage his hands became tremulous and he developed a wrist drop.

Chronologically, the history of sensory changes is rather vague, but he suffered from pains in the arms and back

of the neck, and also numbness in his legs. At the height of his illness he had visual disturbances, was unable to talk, and was incontinent of urine and faeces.

While he did not volunteer the information, later questioning elicited the fact that his urine had been red from about one week after the onset of his abdominal pain. There was no history of the taking of any medicine before admission.

Past History. In August 1948 he had been admitted to this Hospital suffering from abdominal pain and had passed red urine. No diagnosis was made. This was the only previous occasion on which he had had these symptoms and he only noticed that his urine was red on this and on the previous admission.

Physical Examination. He was a thin, intelligent Swazi male of average height. With the exception of the nervous system and skin, all the other systems were normal, and the blood pressure was 130/90.

Central Nervous System. The cranial nerves were all intact.

Upper Limbs. There was a bilateral wrist drop, the following muscles being completely inactive: Extensor carpi radialis longus and brevis, brachioradialis, extensor digiti communis, extensor policis longus and brevis, and abductor pollicis longus. There was weakness of the intrinsic muscles of the hands. All other muscles of the upper limbs were normal.

Sensory Changes. While there was symmetrical analgesia to pin-prick up to both elbows, light touch, joint and vibration senses were normal.

Reflexes. The supinator was absent but the biceps and triceps jerks were brisk and equal.

Lower Limbs. Both legs were thin and very weak, but there was no loss of movement, though tone was reduced.

Sensory Changes. There was analgesia to pin-prick up to the mid-calf on both sides, and a zone of marked hyperaesthesia, starting one inch above Poupart's ligament and extending four inches down the thighs, completely encircling the body. Other forms of sensation were normal.

Reflexes. The knee jerks, ankle jerks, and abdominal reflexes were brisk and equal. The plantar response was flexor.

Skin Changes. There were three small areas of depigmentation, 1-2 mm. in diameter on the wrists, these had apparently been caused by small pimples which had discharged white matter. On the ankles there were five areas of thin skin about 1 cm. in diameter covering old ulcers which had formed spontaneously in 1939.

Psychological Manifestations. No unusual behaviour was noted at any time and consciousness was unclouded up to the end of his life.

Other Investigations. The urine when first seen showed a striking salmon-pink colour and the presence of porphyrin was suspected. Spectroscopic examination showed the presence of metal porphyrin complex and the porphobilinogen reaction was strongly positive. Further examination of the urinary porphyrin is described later. The lead content of the urine was not significantly increased. Blood sugar, cytology and chemistry of the cerebrospinal fluid, were normal and the Wassermann reaction was negative. No abnormal findings were reported on radiological examination of the chest and of the alimentary tract after a barium meal. A blood count showed 13.3 gm. per cent of haemoglobin, 4.1 million erythrocytes per c.mm., mean corpuscular volume 85.3, 6,600 leucocytes per c.mm. Punctate basophilia was not observed.

PROGRESS

Within a few days of admission his urine had lost its characteristic colour, but still showed a faint brownish tinge, and darkened considerably on standing. Improvement was slow but steady, and 10 days after admission the power in his legs had increased considerably. The condition of his arms was unchanged, but the abdominal pain was much reduced. Two weeks later all sensory changes had disappeared. The abdominal pain had practically gone, and he complained only of occasional twinges. He was walking well.

On 24 July, 38 days after admission, he was discharged at his own request. While the wrist drop persisted, there was evidence that the intrinsic muscles of both hands were stronger.

On 8 August he was seen again, and on this occasion there was definite improvement in the condition of his wrists. The brachioradialis, extensor digiti communis, extensor radialis longus and brevis were still paralysed, but the extensor and abductor pollicis longus were acting weakly, and the intrinsic muscles of both hands showed good function.

On 18 October he was re-admitted complaining of abdominal pain, vomiting and pain in the legs. For the previous three days he had been constipated. There was considerable improvement in the power of his arms and legs. The wrist drop had disappeared and detailed examination showed that with the exception of the extensor carpi radialis longus and brevis, all the other muscles were functioning well.

Sensory changes had reappeared in the same form and distribution as before, but were very mild. The urine was red in colour and gave positive chemical and spectroscopic tests for porphyrin.

Over the next 12 days his condition improved considerably but on 30 October he suddenly relapsed. The wrist drop returned overnight with weakness of all his limbs and for the first time phonation became impaired. The original area of hyperaesthesia became intensely painful.

Apart from one brief period of improvement his condition steadily deteriorated. He was always able to move his legs but his arms became paralysed and his voice a faint whisper. The abdominal pain persisted and was only relieved by morphine. Constipation was extreme and repeated enemas were the only means of evacuating the bowel.

Later the diaphragm became paralysed and on 18 November he developed a terminal bronchopneumonia and died on 21 November, five weeks after re-admission.

Post-Mortem Examination. Apart from confirming the presence of the terminal bronchopneumonia there was little of note except recent superficial ulceration of the duodenum. Sections were made of liver, bone marrow, brain and spinal cord but these also showed nothing remarkable.

TREATMENT

There is no known specific treatment for this disease. Abrahams[5] has suggested that Kaolin may be of value as it absorbs porphyrin. This was used during both attacks but as it had no apparent effect during the second its contribution to his recovery on the first is open to question.

Treatment was directed into two channels. Firstly, to relieve abdominal pain, Kaolin, alkalis and belladonna were tried. These were ineffective and Pethidine and morphine had to be substituted. Secondly, in an effort to check the metabolic disorder the following drugs were tried: all the vitamins of the B group including B_{12}, vitamins E and C, crude liver extract, glycine and landrax. As is apparent, no success was obtained with any of the drugs employed.

CHEMICAL EXAMINATION OF THE PORPHYRIN

Direct spectroscopic examination of a 24-hour specimen of urine sent by post from Durban to Johannesburg showed the two absorption bands at 580 mμ : 540 mμ., characteristic of a metal-porphyrin complex; on addition of hydrochloric acid the spectrum reverted immediately to that characteristic of acid porphyrin. A fairly strong band was present at 500 mμ. in both the untreated and acidified specimens of urine. Schlesinger's reaction for urobilin was only very faintly positive whence it is inferred that the 500 mμ. band was mainly due to porphobilin. The porphobilinogen reaction was negative but this was inconclusive in view of the long delay in transit. As stated earlier this reaction was strongly positive on fresh specimens of urine examined in Durban.

Quantitative determinations were made of the coproporphyrin extracted by ether and of the porphyrin adsorbed on a calcium-phosphate precipitate[9]. The 24-hour excretions were: coproporphyrin — 130 γ., phosphate-adsorbed porphyrin 7.7 mg.

Continuous collection was then made for a period of nine days, at first the urine was paler in colour than when the diagnosis was originally made but darkened again towards the end of this period.

On adjusting the pH of the urine to 3.1 no flocculent material separated, but on the addition of alkali a discoloured sediment of calcium salts settled which was separated and sent to Johannesburg for further treatment.

This sediment was centrifuged and the supernatant urine showed no porphyrin on spectroscopic examination after acidification with hydrochloric acid. The precipitate was dissolved in sufficient 2 N hydrochloric acid to convert the porphyrin to the acid form, filtered and the porphyrin flocculated out by adjusting the pH to 3.1. At this and subsequent stages, chromatographic analysis on filter paper strips was performed[10], the results of which are discussed later.

The flocculent material was collected on a filter paper, dried and esterified in methyl alcohol containing 2% sulphuric acid. The ester was recovered in chloroform solution in the usual manner, washed thoroughly with dilute ammonia and several successive quantities of distilled water. On concentrating to small bulk and adding warm methyl alcohol, a moderate crop of crystals separated slowly from a strongly coloured mother liquor. After recrystallization twice from chloroform-methyl alcohol typical fine acicular crystals were obtained which melted at 253-255° C, which corresponds closely to the figure 257-258° C for Waldenström's uroporphyrin III methyl ester.

The mother liquors from all three crystallizations were pooled, dried, moistened with methyl alcohol and an excess of approximately 6 N hydrochloric acid added. After standing at room temperature overnight sodium acetate and sodium hydroxide solutions were added until the mixture was no longer acid to Congo red. One-fifth volume of glacial acetic acid was added and the mixture shaken with two portions of ether which removed part of the porphyrin. Both these fractions were recovered in flocculent form from aqueous solution at pH 3.1 and samples chromatographed. The remainders were esterified but while the ether insoluble fraction yielded crystals similar to the main crop of uroporphyrin ester the ether soluble fraction did not crystallize.

Strip chromatograms were made of the recovered porphyrin before and at different stages during the partition into fractions. Chromatograms of the total porphyrin showed a marked preponderance of uroporphyrin and a smaller quantity of coproporphyrin thus confirming the quantitative determination recorded above. The mother liquor after removal of the uroporphyrin-ester crystals still contained both these porphyrins but the latter was present in relatively much higher concentration.

The two fractions into which the mother liquor pigments were divided by partition with ether showed mainly coproporphyrin in the ether soluble and uroporphyrin in the ether insoluble fraction, but each was contaminated with a trace of the other. All the strips showed faint spots suggesting the presence of very small quantities of porphyrins with three, five, six and seven free-carboxyl groups. Further examination of these could not be carried out as the amount of material available was inadequate.

DISCUSSION

As indicated earlier acute porphyria is not so rare a metabolic anomaly as to be dismissed from clinical consideration. At the time this case was encountered its occurrence in a Negro had not been reported although the diagnosis had been made in a number of cases in white-skinned South Africans. While the investigation of this case was in progress a fatal case in an African Negro student in London was reported[11] and a third presented at the Far East Rand Hospital, Springs, details of which will be reported elsewhere.

Unless the possibility of acute porphyria is in mind, the findings presented by this type of case are very bewildering. The salmon-pink colour of the urine was suggestive and the positive porphobilinogen reaction and spectroscopic evidence of the porphyrin metal complex clinched the diagnosis.

The combination of abdominal colic, wrist drop and porphyrinuria was compatible with lead poisoning. The patient was employed by a jeweller who was interviewed and stated that contact with lead was slight. Further evidence against lead poisoning were the paralysis of the brachioradialis and the sensory changes. The blood film showed no punctate basophilia and subsequent tests of the urine showed the presence of a large amount of uroporphyrin and only a slight increase in coproporphyrin: in lead poisoning the latter is increased and uroporphyrin has not been reported. The normal blood sugar and absence of sugar from the urine precluded diabetic polyneuritis.

The slight skin changes observed were not unlike the healed lesions seen in porphyria cutanea tarda which is not infrequently encountered in South African Negroes. However, a random survey of other patients without porphyrinuria showed that the majority had similar scars on hands and feet which were probably due to past trauma. While the absence of active skin lesions at a time when porphyrin was present in the urine excluded the condition at this stage, this patient may have been of the type that commences with skin lesions and later develops the acute type of porphyria.

All the features present and the history of this case are compatible with acute porphyria and despite its very rare occurrence in Negroes it is presented as such. It is perhaps noteworthy at this stage that whereas acute porphyria is

more common in females than males this and the two other Negro patients referred to were all males.

SUMMARY

The clinical and chemical findings in a fatal case of acute porphyria occurring in a Bantu male have been presented.

No success attended the use of any of the drugs employed.

We wish to express our thanks to Dr. G. A. Drummond and Dr. J. Wainwright for their valuable assistance with this case.

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A FOOD BALANCE SHEET FOR THE UNION OF SOUTH AFRICA

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An attempt is made to balance the needs of South Africa's heterogeneous population against estimated production figures.

The Protein Position. From a protein point of view the major imbalance is caused by two factors, namely

1. Purchasing power.
2. Maldistribution.

TABLE I: DIET SCALES ON WHICH CALCULATIONS ARE BASED (DAILY REQUIREMENTS)

Food Stuffs	*Europeans, Coloureds and Asiatics*	*Urban Bantu*	*Rural Bantu*
Unsifted Wheat Meal ..	6 oz.	4 oz.	—
Maize Meal	4 oz.	12 oz.	16 oz.
Meat, Fish, Eggs ..	6 oz. (as purchased)	4 oz. (as purchased)	4 oz. (as purchased)
Milk	¾ pint	½ pint	½ pint
Fruit and Vegetables ..	16 oz. (as purchased)	8 oz. (as purchased)	10 oz.
Sugar, Jam	3 oz.	3 oz.	1 oz.
Butter, Margarine, Fat	1½ oz.	1 oz.	1 oz.
Pulses	1 oz.	2 oz.	4 oz.

These diet scales are merely illustrative examples of daily requirements in terms of specific foodstuffs, but there are other combinations of foods which would be equally suitable.

Approximately three and a half million natives in the Reserves appear to be getting a very small amount of animal protein, which is made up mainly of animals dying of disease and drought and a small amount of milk which is taken from the cows of the herds of native cattle. Possibly a certain amount of abattoir offal is also consumed by this group.

All these sources of protein do not make up an adequate supply of protein of animal origin. This group, namely the Bantu residing in the Reserves, must receive special attention in connexion with the protein aspect of his diet.

With regard to the Europeans and the better favoured

TABLE II: PER CAPITA DAILY REQUIREMENTS

Nutrients	*Europeans, Coloureds, Asiatics*	*Bantu*
Calories	2,500	2,900
Protein (Grams) ..	66	66
Fat (Grams)	70	65
Calcium (Grams) ..	1	1
Iron (Milligrams) ..	12	12
Thiamin (Milligrams) ..	1·28	1·28
Vitamin C (Milligrams)	36	36
Vitamin A (International Units)	3,250	3,250

TABLE III: ESTIMATED POPULATION AS AT 30 JUNE 1950—BY RACE

Province	European			Native[1]			Asiatic			Coloured		
	M	F	P	M	F	P	M	F	P	M	F	P
Cape ..	458,000	463,000	921,000	11,229,900	1,330,500	2,460,400	9,800	8,300	18,100	461,100	461,800	922,900
Natal ..	134,000	134,000	268,000	856,900	916,100	1,773,000	133,900	127,500	261,400	15,000	13,700	28,700
Transvaal	617,000	601,000	1,218,000	1,949,600	1,454,900	3,404,500	24,000	20,800	44,800	33,000	32,800	65,800
Orange Free State	106,000	103,000	209,000	345,900	362,800	708,700	—	—	—	6,300	6,100	12,400
Union ..	1,315,000	1,301,000	2,616,000	4,282,300	4,064,300	8,346,600	167,700	156,600	324,300	515,400	514,400	1,029,800

Total Population: 12,316,700

[1] Rural Bantu 6,429,400 (of which approximately 3,000,000 reside on European-owned farms).
Urban Bantu 1,917,200
Total Bantu 8,346,600

TABLE IV: TOTAL ANNUAL POPULATION REQUIREMENTS (IN SHORT TONS)

Foodstuffs	*Europeans, Coloureds, Asiatics*	*Urban Bantu*	*Rural Bantu*	*Total*
Unsifted Wheatenmeal	271,704	87,472	—	359,176
Maize Meal	181,136	262,417	1,173,365	1,616,918
Meat, Fish, Egg	271,704	87,472	293,342	652,518
Milk	135,851,859 gal.	43,736,613 gal.	146,670,688 gal.	326,259,166 gal.
Fruit, Vegetables	724,543	174,945	733,353	1,632,841
Sugar, Jam	135,852	65,604	73,335	274,791
Butter, Margarine, Fat	67,926	21,868	73,335	163,129
Pulses	45,284	43,736	293,341	382,361

These estimates are calculated from the diet scales in Table I multiplied by the population numbers in Table III.

TABLE V: TOTAL ANNUAL POPULATION REQUIREMENTS (SHORT TONS[1])

		Europeans, Coloureds and Asiatics	*Urban Bantu*	*Rural Bantu*	*Total All Races*
Protein	Fish and Animal[2] ..	35,142	16,970	56,910	109,022
	Plant	70,283	33,941	113,821	218,045
	Total	105,425	50,911	170,731	327,067
Fat		111,814	50,139	168,144	330,097
Carbohydrates		640,775	395,522	1,326,396	2,362,693
Calories (x 10^6)		3,622,717	2,029,356	6,805,520	12,457,593

[1] Calculated from Daily Requirements standard in Table II x Population figures in Table III.
[2] Taken as one-third of total protein requirements.

TABLE VI: TOTAL AVAILABLE NUTRIENTS 1948—49[8] (CONSUMED 1949)

(*After du Plessis*[1])

1 ton = 2,000 lb. (Short Ton)

Categories	*Protein (Tons)*	*Fat (Tons)*	*Carbohydrates (Tons)*	*Calories (x 10⁶)*
Cereal grains	206,682	71,194	1,475,969	6,683,628
Pulses	10,176	18,822	20,904	266,488
Vegetables	11,560	1,272	63,270	281,030
Sugar products[3]	—	—	529,212	960,952
Fruits	2,311	1,617	13,520	150,451
Animal products[5]	118,055	202,023	52,477	2,157,842
Fish[4]	5,877	2,109	—	38,565
Miscellaneous products[6]	1,553	1,496	15,928	75,724
Other plant oils[2]	—	14,943	—	121,935
Total animal products and fish	123,932	204,132	52,477	2,196,407
Total all plant products[7]	232 282	109,344	2,118,803	8,540,208
Grand Total	356,214	313,476	2,171,280	10,736,615

[1] Physical consumption figures of du Plessis were converted to nutrients by means of factors after Van de Wall.
[2] Includes peanut oil.
[3] Sugar only.
[4] Includes edible marine oil.
[5] Includes edible slaughter fats.
[6] Cocoa and rice only.
[7] Includes miscellaneous products (see [6] above).
[8] This makes allowances for exports, imports animal feeds, etc., and represents the amounts of nutrients which are actually consumed by human beings.

sections of the community, there is an adequate amount of animal protein and with the exception of the very poor members of the community protein inadequacy does not appear to be a problem.

1. *Total Theoretical Requirements.* (a) The nutritional requirements in Table II are based on the recommendations of the National Nutrition Council.

Requirements are calculated according to age, sex and activity of the individual. When dealing with a population or proportions thereof, however, it is possible to calculate such data from per capita 'weighted' requirements. By 'weighted' requirements is meant that an average has been calculated which takes into account the nutritional requirements of the various groups based on age, sex and activity (Table II).

In South Africa, with its heterogeneous population, calculations must be determined by food habits and physiological adaptation. Diets have therefore been worked out for the racial groups. The Europeans, Coloureds and Asiatics are grouped together. The urban and rural Bantu form separate groups.

The diets used as a basis for estimating Bantu requirements furnish approximately the nutrient intake recommended by the National Nutrition Council (Table I).

As it will take time to produce an adequate mixed diet within the reach of all sections of the community, the immediate practical aim should be to enrich, in a suitable physiological balance, maize meal with defatted, doedorized fish meal, which according to reports, could be made available within a reasonably short period.

2. *Actual Amounts Available.* In the following tables data are presented reflecting the available food supplies classified under the main sources. The physical food consumption figures (e.g. tons of maize and wheat and gallons of milk), as given in Table VIII, were converted to protein, fat, carbohydrates and calories by means of suitable conversion factors (Table VI). In Table VII similar data are presented but these data were calculated from a different point of view and in a different manner. Footnotes to each table explain clearly the different methods of calculation.

3. *Estimated Total Shortfall.* With the data from Tables I-VIII available, it was merely a matter of subtraction to obtain the total shortfall of the various products. The results are given in Tables IX-XI. In Table IX the shortfall is given in terms of the various physical commodity units, while in Tables X and XI it is given in terms of protein, fat, carbohydrates and calories. The calculations presented in Tables X and XI are based on the available food supplies as given in Tables VI and VII respectively. The differences between the two methods are explained fully by means of footnotes to each table.

4. *Possibilities of Meeting Shortfall from Purely Physical (Production) Viewpoint.* (a) *Increased Crop Production.*

i. Amongst the legumes the soya bean is nutritionally of the greatest importance on account of its high nutritional value and its quality oil content. It can be cultivated almost anywhere in the summer rainfall areas and used advantageously in rotation with maize. *The main use to which it can be put is the enrichment of maize meal.*

The present annual production of soya beans fluctuates between 13,000 and 20,000 bags. In order to enrich all maize meal to the extent of 3% for human consumption, 450,000 bags are required annually.

To encourage the production of soya bean the price should be adjusted in relation to maize and other oil-bearing seeds.

ii. The production of other legumes, i.e., beans, cowpeas, field peas etc., should not, however, be neglected.

iii. In regard to cereals, the production of maize will

TABLE VII: TOTAL AVAILABLE NUTRIENTS 1948—49[10] (CONSUMED 1949)

(After Van de Wall and Alvord[1])

(1 ton = 2,000 lb.)

Categories	*Protein (Tons)*	*Fat (Tons)*	*Carbohydrates (Tons)*	*Calories (x 10^6)*
Cereal grains	214,864	83,450	1,629,373	7,459,945
Pulses[2]	14,591	13,175	36,387	289,380
Vegetables	8,992	1,013	74,848	312,312
Sugar products[3]	2	—	497,678	893,256
Fruits	2,748	2,427	16,121	196,560
Fish	4,886	853	—	26,208
Animal products[4]	87,819	161,994	35,927	1,646,736
Miscellaneous[5]	5,395	3,787	12,667	96,096
(a) Total animal products and fish	92,705	162,847	35,927	1,672,944
(b) Estimated contributions of abattoir offal[6]	7,570	12,279	—	114,119
(c) Milk consumed in reserves (estimate)[7]	4,249	4,764	5,923	75,600
(d) Meat consumed in reserves (estimate)[8,9]	6,985	10,030	—	94,500
(e) Total animal products including (a), (b) and (c)	104,524	179,890	41,850	1,787,063
(f) Total animal products including (a), (b), (c) and (d)	111,509	189,920	41,850	1,881,563
(g) Total all plant products	241,197	100,039	2,254,407	9,151,453
(h) Grand Total excluding (b), (e) and (d)	339,330	266,700	2,303,000	10,920,000
(i) Grand Total including (b), (c) and (d)	358,101	293,746	2,308,924	11,129,112

[1] Trends projected and assumed that each category contributed the same percentage of the total as in 1945/46.
[2] Including peanut oil and other vegetable oils, locally produced.
[3] Including honey and importation of glucose.
[4] Excluding (i) Milk consumed by natives in reserves.
(ii) Meat consumed by natives in reserves (i.e., animal deaths mainly).
(iii) Edible abattoir offal.
See [6], [7] and [8] below.
[5] Includes imports of miscellaneous products mainly luxury articles and vegetable oils and fats.
[6] Offal estimated as follows:—
(i) Cattle and calves, weighted mean .. 16% of carcase weight.
(ii) Sheep, goats and lambs, weighted mean 17% of carcase weight.
(iii) Pigs, weighted mean .. .. .. 2% of carcase weight.
On this basis estimates made for 1945/46 found that it contributed an extra 8·62% of animal protein, 7·58% animal fat and 6·93% animal calories. These percentages then applied to 1948/49 data.
[7] Estimated as follows:—
Approximately 3½ million cattle owned by natives in reserves. According to Professor Bonsma 41·5% are females, older than 2 years of age. Of them approximately half in milk at a production rate of 50 gallons per cow per year available for human consumption.
Therefore, 41·5% of 3½ million .. 1,452,500
Half in milk .. 726,250
x 50 gal. per cow .. 36,312,500 gal.
It is however doubtful whether all cows in milk are even milked and it would be safe to assume that only two-thirds are milked giving a total production of approximately 25,000,000 gallons available for human consumption.
[8] Estimated by du Plessis at 50,000 tons of beef for 1949. This is probably too high due to an abnormally dry season causing large numbers of livestock to die.
[9] In converting this meat to nutrients, it must be borne in mind that the quality is lower than that of the European-bred animals. These figures given for protein, fat and calories are therefore probably too high, due to the same conversion factors being used.
[10] This makes allowances for exports, imports, animal feeds, etc., and represents the amounts of nutrients which are actually consumed by human beings.

TABLE VIII: ACTUAL ANNUAL AVAILABLE FOODSTUFFS (SHORT TONS)
(After du Plessis)

	1948	1949
Wheat	509,499	501,818
Maize	1,436,800	1,450,827
Other Cereals[1]	119,754	98,563
Meat, Fish, Eggs ..	?	750,131
Milk and Cheese[2] ..	219,190,004	220,688,724
Fruit and Vegetables ..	902,995	1,030,778
Sugar	552,995	529,742
Butter, Margarine, Fat ..	63,916	73,305
Pulses and Oil Seeds ..	39,606	42,548

[1] Including rice. [2] Expressed as gallons of milk.

have to be increased substantially to meet the increased demand. Increased production can be achieved by means of improved agricultural methods and increased efficiency. This will enable the lower income groups, who are dependent upon maize for their calories, to obtain this important basic food at a lower cost.

iv. *Wheat* production should not be encouraged at the expense of the soil, but existing production areas should improve their methods to maintain at least the present production level. It is not advisable to build up a nutritional economy on imported wheat.

v. *Other Cereals.* The only other cereal which is of any importance is kaffir corn. This crop unfortunately does not enjoy the prominence it merits both nutritionally and agriculturally due largely to the disadvantageous price structure.

(b) *Fish.* Defatted and deodorized fish meal must be regarded as one of the best available enrichers of maize meal. It is estimated that to enrich about half the maize meal for human consumption with a 6% addition, approximately 50,000 tons would be required annually.

(c) *Dairy Products.* Dairy products are in very short

TABLE IX: ESTIMATE TOTAL SHORTFALL (SHORT TONS) 1949

	Cereals	*Meat, Fish, Eggs*	*Milk (Gallons)*	*Fruit, Vegetables*	*Sugar, Jam*	*Butter, Margarine and Fat*	*Pulses*
Theoretical Population Requirements.	1,976,094	652,518	326,259,166	1,632,841	274,971	163,129	382,361
Available Foodstuffs[1]	2,051,208	750,131[2]	220,688,724	1,030,778	529,741	73,305	42,548
Shortfall or Surplus	75,114 Surplus	97,613[3] Surplus	105,570,442 Shortfall	602,063 Shortfall	254,770 Surplus	89,824 Shortfall	339,813 Shortfall

[1] After du Plessis.
[2] Includes estimates of meat obtained from animals which have died from disease or drought, and abattoir offals.
[3] Surplus due to inclusion of meat as given in footnote [2] and the particular arbitrary selection of diets shown in Table I.

TABLE X: NUTRIENT BALANCE SHEET FOR 1949 (SHORT TONS)

		Total Available in 1949[1]	*Total Population Requirements*[2]	*Shortfall or Surplus*	*Shortfall or Surplus, as % of Requirements*
Protein	Animal and Fish ..	123,932	109,022	14,910[3] (surplus)	13·68 (surplus)
	Plant	232,282	218,045	14,237 (surplus)	6·53 (surplus)
	Total	356,214	327,067	29,147 (surplus)	8·91 (surplus)
Fat		313,476	330,097	16,621 (shortfall)	5·04 (shortfall)
Carbohydrates		2,171,280	2,362,693	191,413 (shortfall)	8·10 (shortfall)
Calories x (10^6)		10,736,615	12,457,593	1,720,978 (shortfall)	13·81 (shortfall)

[1] After du Plessis.
[2] Calculated from Tables II and III.
[3] Includes estimates of meat obtained from animals which have died from disease or drought, and abattoir offal, and an estimated amount of milk consumed in Native Reserves.

TABLE XI: NUTRIENT BALANCE SHEET FOR 1949 (SHORT TONS)

		Total Available in 1949[1,2]	*Total Population Requirements*	*Shortfall or Surplus*	*Shortfall or Surplus, as % of Requirements*
Protein	Animal and Fish[3,4] ..	104,524	109,022	4,498 (shortfall)	4·3 (shortfall)
	Plant	241,197	218,045	23,152 (surplus)	10·62 (surplus)
	Total	351,116	327,067	24,049 (surplus)	7·49 (surplus)
Fat		283,716	330,097	46,381 (shortfall)	14·05 (shortfall)
Carbohydrates		2,308,924	12,362,693	53,769 (shortfall)	2·86 (shortfall)
Calories (x 10^6)		11,034,612	12,457,593	1,422,981 (shortfall)	11·42 (shortfall)

[1] After van de Wall.
[2] Also includes miscellaneous imports and exports.
[3] Does not include animals which have died from disease or drought, and a more rational estimate of milk consumed in Native Reserves was made.
[4] More in keeping with results of food consumption surveys than corresponding figures given in Table X.

supply, but can be increased considerably with improved pasture management and greater supplementation from arable crops. The price is again of importance and at present levels it is not within the reach of the lower income groups.

5. Best Methods of Ensuring that All Groups Receive Adequate Protein. The best method of ensuring that all groups receive adequate protein is the enrichment of maize meal and wheaten meal with such enrichers as fish meal, soya-bean meal, and calcium.

In order to achieve this it would be necessary to stimulate the production of soya-bean meal.

Increased efficiency and production is the key both to more food and lower costs.

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P. 11

characteristic effects of histamine and is of value clinically in the prevention and relief of many allergic manifestations.

Chlor-Trimeton Injection is specifically indicated where there may be desired a more prompt action than would be obtainable with oral medication—allergies involving the skin and mucuous membranes, urticaria, hives, contact dermatitis, rhus poisoning, bee and other insect stings. *Chlor-Trimeton Injection* may be used in conjunction with desensitization procedures, allowing larger doses of allergen, and producing a higher antigenic titre than would be otherwise possible.

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PASSING EVENTS

MEDICAL PROTECTION

A correspondent has drawn attention to a recent report in *The Lancet* (13 October 1951, p. 681) of the Annual General Meeting of the Medical Protection Society in Great Britain.

In his presidential address on 3 October, Sir Ernest Rock Carling pointed out that the hazards which a doctor had to face had increased. 'Unfortunately Legal Aid had enlarged his liability to litigation. The area committees under the Legal Aid scheme did their best to prevent it being used without good cause, but it was certainly true that people could now bring an action for damages with little risk of loss to themselves. Reports of inquiries by service committees might also contain evidence which could be used by a claimant, sometimes to the detriment of the doctor.'

MEDICAL REGISTER

The attention of the Medical Practitioners whose names appear in the list hereunder is invited to the fact that letters which were addressed to them by the South African Medical and Dental Council to their registered addresses have been returned by the Post Office undelivered. The Practitioners concerned are requested to communicate with the Registrar of the Council, P.O. Box 205, Pretoria without delay advising him of their correct addresses for registration purposes. Failure to do so may result in the erasure of their names from the register.

Elk, Sydney;
Ferguson, Ann;
Guild, William John Forbes;
Sanders, Eric John;
Southey, Philippa Evelyn Isabel Metcalf;
van der Merwe, Ivan;
Wardill, William Edward Mandall;
Ridsdel, William Bramwell.

MOTOR CAR INSURANCE FOR MEMBERS OF THE MEDICAL ASSOCIATION

Members are reminded that it will be to their benefit to reply to the circular recently forwarded to them with a newsletter. Doctors using their cars for professional purposes can effect a considerable saving in the premium to be paid by communicating with Messrs. Edward Lumley and Sons, P.O. Box 4639, Cape Town, the Association's official insurers for this class of motor-car insurance.

REVIEWS OF BOOKS

HUMAN PHYSIOLOGY

Human Physiology. By Bernado A. Houssay, M.D. (Pp. 1,118 + xvi, with 499 illustrations. $14.00.) New York: Toronto: London: McGraw-Hill Book Company, Inc. 1951.

Contents: 1. The Internal Environment and the Blood. 2. The Circulation. 3. Respiration. 4. Digestion. 5. Metabolism and Nutrition. 6. Internal Secretions. 7. Reproduction. 8. The Formation and Excretion of Urine. 9. The Nervous System. Index.

There are few textbooks of physiology in the English language which are so comprehensive that they satisfy the needs of the physician as well as of the undergraduate medical student. To this small and distinguished collection has been added another peer in the shape of the first edition in English of one of the more remarkable accounts of human physiology produced by Prof. B. A. Houssay and his team of eminent collaborators.

Professor Houssay is, of course, well known for his remarkable contributions to many branches of physiology, having been awarded the Nobel prize for medicine in 1947.

The present volume has already achieved distinction amongst South American medical students, and the present translation has given the authors an opportunity to bring out what is virtually a completely revised issue.

Professor Houssay rightly stresses in his preface that the primary emphasis in the approach to physiology should be comparative but he recognizes the need to integrate this approach with the physiological basis of clinical medicine which, after all, often conducts instructive experiments that throw much light on the normal functions of the body. It is not so easy to agree with his postulate that a utilitarian approach to scientific knowledge and research 'ends in a narrow rut of mediocrity'. The most challenging example to his thesis is the project which led to the successful explosion of an atomic bomb.

The authors' stand-point, however, does not vitiate the extraordinarily encyclopaedic account of the normal functions of the human body. As his distinguished team of co-authors guarantees the data presented are extremely up to date and the team-work in writing has made the volume authoritative in all its parts. The illustrations are numerous and illuminating.

The volume should be found particularly interesting and helpful to physicians as well as to students. It is an admirable work of reference.

CROSS INFECTION IN HOSPITALS

The Control of Cross Infection in Hospitals. By the Cross Infection in Hospitals Committee of the Medical Research Council. (Pp. 49, with figures. 1s. 9d.) London: His Majesty's Stationery Office. 1951.

Contents: Introduction. 1. Prevalence and Consequences of Cross Infection. 2. Sources and Modes of Infection. 3. The Prevention and Control of Cross Infection. 4. Procedure Following the Occurrence of Infection in a Ward. Appendices: A. Disinfection and Sterilization. B. Rules for Isolation Nursing in Cell or Open Ward. C. Special Precautions for Maternity Units. D. Rules for a Ward Dressing Team. E. Application of Dust-Laying Oils to Floors, Blankets and Linen. F. Course of Practical Bacteriology for Nurses. References.

Cross infection is still only too common in many of our hospitals. These hospital infections are very largely avoidable. The avoidance does, however, require an accurate knowledge of the sources and modes of infection and of the methods of control. The best remedy often lies in hospital construction, but even in an ideal hospital the dangers of the spread of infection cannot be eliminated completely.

Ignorance of even the most elementary rules for the prevention of hospital infection is deplorably common. For this reason it was found necessary for the Medical Research Council to publish the first edition of this memorandum during the early years of the war. The new addition is equally welcome and once again focuses attention on this important subject.

The booklet is authoritative, concise and accurate. It should be read by every doctor, medical student and nurse. Its perusal could well be made compulsory for every member of the professional and nursing staff of the larger hospitals.

IMMUNOLOGY

Immunology. By Noble Pierce Sherwood, Ph.D., M.D., F.A.C.P. (Pp. 731, with 21 figures and seven plates (in colour). 3rd ed. 68s.) St. Louis: The C. V. Mosby Company. 1951.

Contents: 1. Infection and Infectious Agents. 2. Host-Parasite Relationship. 3. Inflammation and Leucocyte Response. 4. Anatomical and Physiological Factors in Infection and Resistance of the Individual. 5. The Reticulo-Endothelial System. 6. Natural and Acquired Immunity. 7. Immunity Mechanisms in Experimental Infections. 8. Natural and Immune Antibodies. 9. Complement. 10. Isohemagglutinins—Blood Groups. 11. The Rh and Hr Factors of Human Blood. 12. Nature, Formation and Action of Antibodies. 13. Measurement of Antibodies. 14. Mechanism of Antigen-Antibody Reactions, Cellular Agglutination. 15. Precipitins. 16. Toxins and Antitoxins. 17. Normal, Convalescent and Immune Sera. 18. Serum Reactions. 19. Biological and Antigenic Specificity. 20. Modified and Conjugated Antigens. 21. Bacterial Antigens and Specificity. 22. Recapitulation of Chapters on Specificity. 23. The Importance of Antibodies in Diagnosis. 24. The basis of Bacterial Complement Fixation Technique. 25. Complement Fixation in Syphilis. 26. Flocculation Tests in Syphilis. 27. Hypersensitiveness. 28. Hypersensitiveness due to Infection. 29. The Significance of Allergy in Tuberculosis and a few other Diseases. 30. Hypersensitiveness, Human Idiosyncrasies. Appendix.

This is the third edition of a book originally written for medical students. The new edition includes a new chapter on Rh, and other parts of the book have been rewritten. It is a comprehensive treatise not only of immunology but of the related branches of medicine which have a bearing on purely immunological phenomena. It deals, in fact, with a wide range of host-parasite relationships such as phage and bacteria on the one hand, and the human host and its pathogens on the other.

As is almost inevitable in a book with such a wide scope some subjects are dealt with very briefly. Little fault can be found with the purely immunological aspects of the book. It is perhaps unfortunate that, for instance, the immunochemical work of Harington and his co-workers receives but scant attention. An occasional reference given in the text has been omitted from the list of references at the end of the chapter, e.g. Anderson (1946) in Chapter IV, and an occasional name is spelt incorrectly (e.g. Mesrobeno, Chapter I).

It is an advantage from the student's point of view that definitions and descriptions are given freely. They are widely scattered through the text and as a result an impression is obtained of a lack of continuity. This does not make for pleasant reading.

The book is otherwise well written and it is well documented. It is without doubt a valuable book, particularly for students and teachers.

ORTHOPAEDIC EQUIPMENT

Instruments and Apparatus in Orthopaedic Surgery. By E. J. Nangle, M.B., Ch.B., F.R.C.S. (Pp. 231, with 200 figures. 42s.) Oxford: Blackwell Scientific Publications. 1951.

Contents: The Principles of Splinting. 2. Principles of Splinting in Poliomyelitis. 3. Mechanical Respirators employed in the Treatment of Poliomyelitis. 4. Plaster of Paris Technique. 5. Orthopaedic Frames. 6. Counter-Weighted Appliances. 7. Arm Splints. 8. The Spinal Brace. 9. Calipers. 10. Hip Splints. 11. Denis Browne Talipes Splints. 12. Foot Appliances. 13. Surgical Boots. 14. Pylons. 15. Plastics. Authors' Index. Subject Index.

Quot homines tot sententiae may be used with particular reference to orthopaedic appliances, but in this book the author has specifically devoted himself to explaining the principles and the materials upon which the various appliances are built, and has illustrated these with a particular type of well-tried model. In the words of Bruce Gill, one should 'study principle rather than methods. A mind that grasps principles will devise its own methods'. The reader of this work will obtain a clear idea of the requirements of an appliance for any particular region.

The chapter on counter-weighted appliances is excellent and gives voice to Bunnell's dictum that one should 'splint to mobilize'. The affected part is kept well splinted, while the rest of the patient is permitted to be as active as possible in the prevailing circumstances.

The section on plastics, written by Dr. Scales, sign-posts the modern trend for the use of lighter splints and appliances. Much has already been done in this direction, but much remains to be done before plastics will supplant the materials at present in common use. Dr. Scales provides much information about the structure and uses of the various plastics and indicates some of the difficulties associated with their use.

This book is well produced and illustrated; the print and paper are excellent. There is one small error on page 178, where the text should refer to Fig. 167 (not Fig. 176).

The book can be strongly recommended to all orthopaedic surgeons, and students of orthopaedic surgery. It will serve as a book of reference for many years to come.

ORTHOPAEDIC NURSING

Orthopaedic Nursing. By Mary Powell, S.N.R., M.C.S.P. (Pp. 400 + xii, with 210 photographs. 25s.) Edinburgh: E. & S. Livingstone Limited. 1951.

Contents: 1. An Introduction to Orthopaedic Nursing. 2. General Principles of Orthopaedic Treatment. 3. Physiotherapy in Relation to Orthopaedics. 4. Plaster of Paris Technique. 5. Splints and Appliances. 6. Congenital Deformities. 7. Deformities of the Spine. 8. Deformities of the Legs and Feet. 9. Tuberculosis of the Bones and Joints. 10. Tuberculosis of the Spine. 11. Tuberculosis of the Hip Joint. 12. Tuberculosis of the Knee Joint. 13. Tuberculosis of Other Bones and Joints. 14. Acute Haematogenous Osteomyelitis. 15. Pyogenic Arthritis. 16. Osteo-Arthritis. 17. Rheumatoid Arthritis. 18. Spondylitis Ankylopoetica. 19. Lumbago and Sciatica. 20. Epiphyseal Lesions. 21. Rickets. 22. Acute Anterior Poliomyletitis. 23. Spastic Paralysis. 24. Injuries of Bones and Joints. 25. Injuries of the Upper Limb. 26. Injuries of the Lower Limb. 27. Injuries of the Spine, Ribs and Pelvis. 28. Birth Fractures and Compound Fractures. 29. Peripheral Nerve Lesions. 30. After-Care (By Mona Williams). Index.

This book exudes the very spirit of Oswestry and is written by one who is obviously a great teacher as well as a great exponent of the principles and art of orthopaedic nursing. 'The proper treatment of the orthopaedic case lies not only in the care of the body, but in the creation of an atmosphere of hope, of happiness, of security and homeliness.' Herein lies the secret of the successful nursing of the long-term orthopaedic case.

With its precise descriptions of the nursing care of the various types of cases, this book will provide the nurse with a more than adequate knowledge of the difficulties to be encountered and how to resolve them.

The chapters on the care of patients with tuberculous bones and joints are excellent and should be read not only by nurses, but by medical students and post-graduate orthopaedic students. It is, however, invidious to choose any particular chapters for comment, in a book of such uniform excellence.

Here is another winner for the publishing house of E. & S. Livingstone, and it is up to the usual high standard of their other publications.

THE COMPOSITION OF PROTEINS

The Amino Acid Composition of Proteins and Foods. By Richard J. Block, Ph.D., and Diana Bolling, B.S. (Pp. 576 + xxxviii, with XI Tables. Second Edition. 75s. 6d.) Oxford: Blackwell Scientific Publications. United States of America: Charles C. Thomas. 1951.

Contents: 1. The Diamino Acids Arginine, Histidine, Lysine, Hydroxylysine, and Citrulline. 2. The Aromatic Amino Acids Tyrosine, Tryptophane, Phenylalanine, Diiodotyrosine, Thyroxine, and Dihydroxyphenylalanine. 3. The Sulphur-Containing Amino Acids Cystine, Cysteine, and Methionine. 4. The α-Hydroxy Amino Acids Serine and Threonine. 5. The 'Leucines' Leucine, Isoleucine, and Valine. 6. The Dicarboxylic Amino Acids Aspartic Acid and Glutamic Acid. 7. Glycine and Alanine. 8. Proline and Hydroxyproline. 9. General Methods. 10. Summary Tables. 11. Protein and Amino Acid Consumption in the United States. Bibliography. Subject Index.

The nutritive importance of proteins in animal and plant life is well known. It is also recognized that there is a world shortage of protein and one of the problems of nutritionists everywhere is to find ways and means of making more protein available for protein-hungry populations.

The object of this work, as the authors say, is to present to the average reader the widely scattered literature on the methods and results of protein analysis in the most easily usable form. This they have succeeded in doing and the work should be of great value to biochemists, food chemists and nutritionists. The average medical reader, however, will find it of too technical a nature to help him in the elucidation of the protein problem.

CORRESPONDENCE

RULES OF PRACTICE

To the Editor: It is encouraging to find that at least one Branch of the Medical Association of South Africa, viz. the Cape Western Branch, has seen fit to appoint a Special Committee to consider rules of practice applicable to general practitioners and specialists. Time is overdue to consider this matter as the present unsatisfactory conditions of general and special practice require to be placed on a more recognized and ethical basis.

The standard of conduct that should be observed by both general practitioners and specialists must admit of no relaxation one way or another. The special committee recommend that: 1. 'The (general) practitioner should not undertake treatment and diagnostic procedures which by recognized custom fall within the domain of a specialist, etc.'

These 'diagnostic procedures', I take it, cover such things as electrocardiograms. As matters stand every G.P. these days is arming himself with an electrocardiograph, an instrument which although almost foolproof in simplicity of operation is, nevertheless, one of the most difficult aids to diagnosis to interpret.

Then again, many G.P.'s are now doing their own artificial pneumothoraces. This therapeutic procedure, easy enough to perform, requires much careful thought and special experience if one is to avoid the tragedies that often follow its indiscriminate use.

So much for the general practitioner; now for the specialist. The latter should not see patients except through their own doctor; they should, in deed and in fact, be true consultants, and where a patient is referred to them for an opinion, the specialist should in no circumstances, either then or later, take on that patient for treatment unless specially asked to do so by the practitioner in attendance. The other recommendations by the Special Committee are only those which any well-ordered and self-respecting medical community usually follows.

The special committee, suggests, *inter alia*, that the maximum fee for general practitioners should be raised, but how this is going to help them (the G.P.'s) to maintain a stricter ethical standard I fail to see. Already in some localities the general practitioners are allowed to charge a fee of £2 12s. 6d. for an E.C.G. over and above their ordinary visiting fee. The increased fee for an E.C.G. appears merely to have whetted their appetite for still more E.C.G.'s.

Vincent P. Norman.

C.N.A. Building,
East London.
15 November 1951.

THE 'TICKEY-A-WEEK DOCTOR'

To the Editor: Dr. J. H. L. Shapiro's letter about *The Tickey-a-Week Doctor* in the *Journal* of 13 October 1951 certainly makes one wonder what is happening to the medical profession. Strange though it may seem, doctors and their families need money with which to buy food, clothing and shelter. High ideals may nourish the soul but they can do little to support the body.

Dr. Shapiro has stressed the moral aspect of the case. Let me deal with the economic side of a doctor serving a tickey-a-week patient and being at his beck and call 24 hours a day, seven days a week, and 365 days a year, including Christmas and all other public holidays.

Three-pence a week means three-sevenths or about half-a-penny a day. With a day's earnings the doctor will not be able to buy anything. However, two days' pay will enable him to buy a small box of matches or an 'all-day sucker'.

With a week's pay, he can buy a small apple or a bunch of carrots or a razor blade. Two weeks or 336 hours on duty will enable him to buy 10 cigarettes. A month's service or 720 hours of duty will allow him to purchase one mutton chop or a loaf of bread plus half-a-pint of milk. Two months' work (1,440 hours) will provide him with the fee for a haircut.

With 11 weeks' pay he can buy 1 lb. of butter while 4 months' fees will enable him to buy 1 lb. of mutton, at its present-day price.

After 6 months, he will have earned enough to buy 1 lb. of tea, and after a year 1 chicken, with which to celebrate Christmas.

In regard to clothing, he must work 10 weeks in order to buy a handkerchief, 32 weeks for a pair of socks, 3 years for a shirt, and 8 years for a pair of grey flannel trousers.

By keeping his nose to the grindstone for 20 years, he will be able to buy himself one of the less-expensive types of summer suits. After 40 years, or three-and-a-half million hours on duty, he will be able to have his tailor make him a suit—a not too expensive one.

If he is a superman and can work for 80 years, he will have earned enough to provide for a coffin and funeral expenses after his happy sojourn on earth. However, the tickey-a-week doctor cannot afford to stop working so soon. There is still his car (£900) to be paid for. To do this he must work for 1,384 years.

With these few comments, may I wish the tickey-a-week doctor health, prosperity and a long life.

General Practitioner.

16 November 1951.

PRESCRIPTION AND DISPENSING

To the Editor: May I crave a small space in your valuable *Journal* to correct a few opinions so frequently expressed in the correspondence columns of late in the *Rand Daily Mail* about doctors, dispensing and chemists.

1. Doctors, in by far the larger number of cases, do not charge £1 1s. a visit; the fee, if they ever get paid, is 12s. 6d. to 15s. a visit and much less for non-Europeans.

2. Doctors do not as a rule dispense; they do so only for non-Europeans and then charge considerably less for the medicine than do chemists.

3. Now what about the other side: the ills and wrongs committed by chemists—substitutions and counter-prescribing are rife.

I have even been told, on occasions, of chemists who do not hesitate to take a patient into a back room to give injections, pull teeth, etc.

M.B., Ch.B.

Pretoria.
18 November 1951.

THE ART OF CONSULTATION

To the Editor: Few will dispute with that outstanding teacher, J. C. Spence, that 'the essential unit of medical practice, is the occasion when, in the intimacy of the consulting room or sick room, a person who is ill or believes himself to be ill, seeks the advice of a doctor whom he trusts. This is a consultation, and all else in the practice of medicine derives from it. The purpose of a consultation is that the doctor, having gathered his evidence, shall give explanation and advice'.

Most doctors acquire the art of consultation, to a lesser or greater degree, in the hard school of experience. However, a student should be able to learn from his teachers, and it will be agreed that it is in the Out-Patient Department, that this approach can be most successfully demonstrated.

Unfortunately, existing conditions are not always favourable. In many hospitals, overcrowding presents a serious problem. It is not unusual for a group consisting of doctor, students and attending nurse, to be crowded into a small cubicle; sometimes two or three such groups may be working in one room, separated only by a screen or make-shift curtain. It is obvious that no real consultation is possible where so little privacy exists.

Another problem is concerned with the choice of staff for teaching purposes. An experienced teacher tends to concentrate on the everyday problems of medical practice, while the less experienced one is likely to pay more attention to rare or obscure conditions.

What can be done to remedy this state of affairs? When I was in England last year, I was greatly impressed by the method of Out-Patient teaching introduced by Prof. J. C.

Spence in his paediatric department at Newcastle-on-Tyne. A number of small rooms free of benches and blackboards, and simply furnished, is provided. The consultations are given by the most experienced members of the staff. 'Two or three students are allowed to watch and listen with as much self-effacement as possible. They observe the greeting. They hear the questioning. They watch the examination, and finally they listen to the explanation and advice. They do not examine the patients. When the students have accepted this role they appear to appreciate the occasion. They share in the sense of relief when the explanation removes brooding fears. They may even feel a little of the anguish when a tragedy unfolds itself. After the patient has left the room, discussion centres on the consultation while the pathology and diagnosis of the disease is commented on only briefly. The choice of words used in explanation is reviewed. The restraint of words in giving advice is recalled. An experienced teacher using this instrument can thereby reveal to a student a great deal about individuals and about life itself, which cannot otherwise be seen in hospitals. I have mentioned the paradox of being unable satisfactorily to demonstrate the art of consultation in the presence of a third person. The students are made aware of this in a preliminary talk. They are told that before explanation and advice can be given to a patient they must make three diagnoses—the diagnosis of the disease, the diagnosis of the concept or fears of the disease in the mind of patient or parents, and thirdly the diagnosis of the patient's capacity to understand the explanation and follow the advice. They are told also that it is the patients' privilege to be interviewed without students being present if they so desire it. Less than 10% demand this privilege, and for them it is readily provided.'*

Having had the privilege of seeing these principles applied, I am convinced they represent a great advance in medical education, and their importance should be more deeply appreciated.

*Spence, J. C. (1949): *From the* Rep. Conf. Mental Health, London: National Association for Mental Health, 39 Queen Anne Street, W.1.

I. Mirvish.

Commercial Buildings,
90 St. George's Street,
Cape Town.
19 November 1951.

SICKLE CELL ANAEMIA

To the Editor: I have read with interest the report published by Grek and Findlay[1] in which they describe a patient with sickle cell anaemia. They point out some of the anomalous features of the case, e.g. severe destruction of bone localized to one or two vertebrae, marked narrowing of some of the intervertebral spaces with irregularity and blurring of the adjacent surfaces of the vertebral bodies, and the absence of ulcers on the legs. There is another feature worthy of discussion and this is the absence of sickling in the blood of the patient's father and brother.

Neel[2, 3] and Beet[4] have both advanced the hypothesis that sickle cell anaemia represents the homozygous condition, whereas the sickle cell trait will occur in heterozygous individuals. Of 94 parents of children with sickle cell anaemia examined by Neel,[3] all but one were found to have the sickle cell trait. Hence the interest in the absence of sickling in the blood of the father of Grek and Findlay's patient. The diagnosis of sickle cell anaemia is not necessarily invalidated by Neel's observations, since one apparent exception remained unexplained. Furthermore, Singer[5] reported two families in which the blood of the mothers of children with severe sickle cell anaemia did not show the sickling phenomenon even when the haemoglobin of their red cells was completely reduced by various procedures. It has also been suggested that an individual heterozygous for both the thalassaemia and sickling genes may present a picture clinically indistinguishable from sickle cell disease.[6] It is, nevertheless, uncommon to find an unaffected parent, so that it might be worth doing other investigations. The white cell count might be repeated since absence of leucocytosis is not very common, the reticulocyte count could be done and the red cell fragility determined. Further, since the type of sickling in the two conditions is different,[3, 7] a report on the type of sickling observed would be helpful, as would an investigation into the presence or absence of abnormal haemoglobin.[8, 9] The father could be re-examined and both the patient and her father could be genotyped in order to eliminate a discrepancy between the legal and biological parentage. Other techniques for eliciting sickling might be attempted on the father's blood.

If these facts could be established adequately the case might assume even greater importance.

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C. Merskey.

Department of Medicine,
University of Cape Town.
21 November 1951.

ETIOLOGY OF CANCER

To the Editor: It seems strange that recently you devoted an uncritical editorial (this *Journal*, 11 August 1951) to a prolix masterpiece of questionable validity by local authors and have, as yet, made little if any mention of another work by an illustrious South African which has been enthusiastically reviewed by almost every reputable journal in the English-speaking world.* I refer, of course, to Charles Berman's *Primary Carcinoma of the Liver* (1951). This scholarly study has focussed world-wide attention on a problem peculiar to Africa—to such an extent that the British Empire Cancer Campaign is now considering 'an expedition . . .' to Africa 'in search of the cause of cancer' (*Lancet*, 20 October 1951). The fact that the BECC considers such action necessary is a sad reflection on the miserable lack of facilities and funds for those of us in South Africa who are capable of exploiting this wealth of material.

However, there are aspects of the problem which your *Journal* should certainly publicize. A recent article by Cook, Duffy and Schoental (Brit. J. Cancer, 1950, **4**, 405) showed that the locally prevalent herb *Senecio jacobea* produces carcinoma of the liver in rats, and suggested that this might be an etiological factor in primary liver cancer in the Bantu. For some time there has been a glib assumption that this tumour is caused by a nutritional deficiency, but all attempts to prove this experimentally have failed. Actually, an additional insult in the form of a toxic 'carcinogen' is essential. The indigenous herbal medicines widely consumed in Africa include vegetable drugs such as the aforementioned Senecio, as well as many unknown irritants. The Bantu Pharmacopeia may well include colchicine and podophyllin-like substances, which are known mitotic poisons, and possibly also mutagens and carcinogens. It is quite conceivable that one or more of these is causing carcinoma of the liver in the malnourished African.

It behoves your *Journal* to make an issue of this and arouse the interest of those South African doctors who may be in a position, by virtue of their proximity to African herbalists and the confidence they inspire in tribal authorities, to obtain this vital information.

Annabelle Cohen, B.A., M.Sc. (New York).

Experimental Oncology Laboratory,
Radiation Therapy Department,
Johannesburg General Hospital.
22 November 1951.

[* This monograph was reviewed very fully (but not very enthusiastically) in this *Journal* on 8 December 1951, p. 918. Our correspondent was not aware of this at the time her letter was written.—*Editor*.]

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The SPEED you need is yours when film, screens, and chemicals bear the Kodak label. Then, because these products are made to work together, the radiographer is assured the utmost in speed in every step, from initial exposure to final processing . . . and the maximum diagnostic value.

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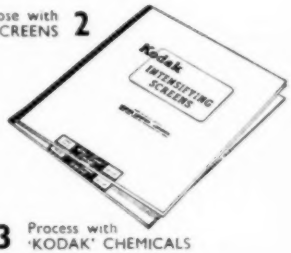
Expose with 'KODAK' SCREENS 2



KODAK PRODUCTS FOR RADIOGRAPHY

Blue Brand and 'Kodirex' X-ray Films . . . 'Flurodak' and 'Fluropan' Films for mass miniature radiography . . . High Definition and Ultra Speed X-ray Intensifying Screens . . . Exposure Holders . . . X-ray Developers, Developer-Replenishers and Fixers . . . Processing Units and Drying Cabinets . . . Safelight Lamps . . . Hangers, Thermometers . . . Film Corner Cutters . . . Illuminators.

3 Process with 'KODAK' CHEMICALS

KODAK (South Africa) Limited

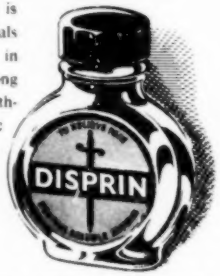
CAPE TOWN · JOHANNESBURG · DURBAN

'KODAK' is a registered trade mark.

ASPIRIN ***is an acidic substance — sparingly soluble***

DISPRIN ***is substantially neutral, stable, soluble and palatable — and in solution forms calcium aspirin***

The reasons for preferring calcium aspirin to aspirin lie chiefly in the fact that it is a neutral, soluble and bland compound, whereas aspirin is acidic, sparingly soluble and may act as a gastric irritant. But calcium aspirin has a defect of its own—chemical instability; and in consequence, attempts to manufacture it in the form of tablets that could be depended upon to remain free of nauseous breakdown products, under reasonable conditions of storage, have hitherto met with little success. These difficulties have now been overcome. Disprin, a stable tablet preparation, readily dissolves to yield a palatable and far less acid solution of calcium aspirin that can be prescribed in all conditions in which acetylsalicylate administration is indicated. Extended clinical trials have shown that Disprin in massive dosage, even over long periods, has been tolerated without the development of gastric or systemic disturbances except in cases of extreme hypersensitivity.



Clinical samples and literature supplied on application.
Special hospital pack — prices on application.

Made by the manufacturers of "Dettol"

RECKITT AND COLMAN (AFRICA) LTD., P.O. BOX 1097, CAPE TOWN

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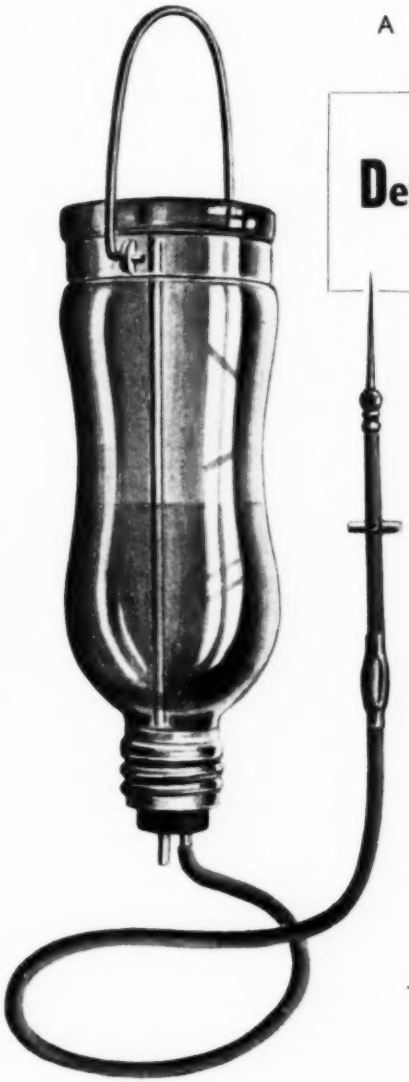
A FURTHER DEVELOPMENT



Dextran-Benger 10%★

Following the findings of various workers* on sodium-free dextran in the treatment of nephrotic oedema and the toxaemia of late pregnancy, Dextran-Benger 10% is now available in South Africa for clinical work.

There appears to be an increasing body of opinion that a NaCl-free plasma substitute may be used with great advantage when transfusion fluids containing sodium ions are contra-indicated.

Dextran-Benger 10% has all the advantages of the Dextran-Benger now in routine use. In addition the absence of sodium chloride widens the range of usefulness of dextran solutions in blood volume replacement.

PAARVO VARA—Acta. Obst. et Gyn. Scand. 1950 xxx July 6.

G WALLENIUS—Scand. J. of Clin. & Lab. Inv. 1950. 2.228.

★ *Full literature is available on request from*
MESSRS. BRITISH CHEMICALS & BIOLOGICALS, (S.A.) (PTY.) LTD.
P.O. Box 5788, JOHANNESBURG.

THIS SIGN IS YOUR SAFEGUARD

When selecting an insulin, doctors in all parts of the world have long preferred to signify Insulin A.B., realising that the sign "A.B." is a guarantee of excellence. It is a safeguard for both doctor and patient. The preference for Insulin A.B. is based on trust and experience—on the knowledge that the sign "A.B." ensures all that can be desired in quality and performance.

INSULIN A.B.

INSULIN A.B.
Globin Insulin (with zinc) A.B.
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The Medical Association of South Africa
Die Mediese Vereniging van Suid-Afrika

AGENCY DEPARTMENT : AGENTSKAP AFDELING

JOHANNESBURG

Medical House, 5 Esselen Street. Telephone 44-9134-5, 44-0817
Mediese Huis, Esselenstraat 5. Telefone 44-9134-5, 44-0817

PRAKTYKE TE KOOP : PRACTICES FOR SALE

(Pr/S32) Johannesburg practice. Average annual gross receipts £1,500. Premium of £1,250 includes surgery equipment and furniture.
(P/O9) Southern Rhodesia, partner required, Englishman preferred.
(Pr/S34) Progressive Transvaal town dispensing practice. Average gross income £3,500 p.a. Excellent surgical facilities. Owner going overseas.

MEDICAL EQUIPMENT

(I/O19) Zeiss microscope. Condition as new. £55.
(I/O23) Heavy based Irrigator stand, height adjustable, complete with glass flask and hook to carry vacolitre flasks. £7.
(I/O24) Bausch & Lomb microscope. Condition as new. Oil, high and low power lenses. Two eye-pieces. £60.
(I/O25) Instrument cabinet, £7 10s.
(I/O26) B.G.E. "Hanovia" Ultraviolet lamp. Good condition. £25.
(I/O27) Examination couch, £7 10s.

* * * *

KAAPSTAD : CAPE TOWN

Posbus 643, Telefoon 2-6177 : P.O. Box 643, Telephone 2-6177

PRAKTYKE TE KOOP : PRACTICES FOR SALE

(821) Eastern Province hospital town. Gross cash receipts £2,200. Premium required £850 which includes drugs, furniture and instruments valued at £350. Mainly non-European at present but with definite scope for future.
(673) Natal. Average gross receipt £1,650 p.a. Prescribing. Premium required £1,275. One appointment £200 p.a. Good scope for expansion. Double-storied seven-roomed house situated on 1½ acres and separate surgery building for sale at £6,500. Surgery may possibly be rented by arrangement at approx. £8 p.m. Picturesque surroundings. Climate notably cooler than that of the coast.
(746) Large dispensing practice, mainly non-European. Average annual cash receipts approx. £5,200. £5,500 required for premium, drugs and surgery furniture. Details on application.
(350) Eastern Cape Hospital town. Total gross receipts preceding 13 months £3,700. One appointment. Premium £1,500. House for sale at £3,000. Large bond available. £700 rebate if appointment not transferred. Practice offers great scope for practitioner with surgical ability.
(848) Cape Town, Southern Suburbs. Solus practice. Average annual receipts £2,088. Premium and terms open to discussion. Excellent opportunity.
(895) Partnership share in specialist physicians' practice. Details on application.
(644) Durban Central. Mainly Indian and Native cash practice. Average annual gross income £1,235. Premium of £500 required for goodwill, inclusive of furniture and fittings and drugs. Terms may be arranged.
(915) Western Province hospital town. House and surgery to let. Excellent opportunity for doctor capable of doing major surgery. Details on application.

VENNOOTSKAP/VERLANG : PARTNERSHIP REQUIRED

(811) Partnership share in Cape or Natal in predominantly English-speaking practice with min. net income £2,500 p.a.

ASSISTENTE/PLAASVERVANGERS VERLANG ASSISTANTS/LOCUMS REQUIRED

(903) Karoo hospital town. As soon as possible until end January. Salary to be arranged. (Quote also 902.)

MEDICAL EQUIPMENT FOR SALE

(772) Strand. C.P. Couch, instrument and dressing tables, cupboards and waiting-room furniture, at approx. £100. Instruments at £100.
(758) Electrocardiograph. Sanborne Cardiette. Weight 24 lb. Perfect working condition. Used by Cape Town specialist physician. £160 or nearest offer.
(674) *British Encyclopaedia of Medical Practice.*
(878) White wooden cabinet for surgery. Five feet high. Top half glass doors and shelves. £23 10s.
(909) Slit Nitra Lamp (Prof. Guilstrand's). Good working order. £20 or nearest offer.

CONSULTING ROOMS REQUIRED

(907) Cape Town. Two rooms and share waiting room and services of nurse/receptionist. Urgent.

* * * *

DURBAN

112 Medical Centre, Field Street, Telephone 2-4049

PRACTICES FOR SALE

(D1) In large coastal town suitable for a F.R.C.S. or M.R.C.O.G. Total gross receipts from June 1950 to June 1951, £4,995. Premium £3,100 includes drugs, fittings, surgery furniture and instruments. Terms could be arranged with reasonable cash deposit. Owing to ill-health owner wishes to sell immediately.
(D2) Zululand practice, 136 miles from Durban. Premium £3,000 includes drugs and instruments. Gross receipts for 1950, £4,881. This is a dispensing practice in a rural area; the nearest hospital is 23 miles away. Four appointments totalling £720 per annum are held and, it is believed, are transferable. House is also for sale.
(D3) In large coastal town. Prescribing practice. Premium required £1,660. Total gross receipts 1950, £1,611. From June 1950 to June 1951, £1,761. Present owner wishes to retire to the country.

ASSISTANTS REQUIRED

(DA1) Assistant required in Natal country practice as soon as possible for at least one year. Single man preferred. General practice, 75% Native. Would suit recently qualified man who wants experience in G.P. and at the same time three–four hours for afternoon study. Very little surgery or midwifery, at present sent to Pietermaritzburg. Salary £950-£1,000 per annum depending on experience.

P.U.T.C. Staff Sick Fund

Applications are invited from qualified medical practitioners for the position of 'Part-time Medical Officer' for the Pretoria Branch of the above Fund.

The number of Members to be cared for is as follows:—
Europeans: Married 39; Single 15.
Non-Europeans: 346.
Remuneration will be at the rate of:—
Europeans: 22s. 6d. per annum single members.
£3 10s. per annum married members.
Non-Europeans: 14s. per annum per member.
A transport allowance of £10 per month will be payable.

Applications should be addressed to Honorary Secretary, P.O. Bramley, Johannesburg and must be received by 15 January 1952. The successful applicant would be required to commence duties as early as possible after appointment.

[This appointment has been approved by the Southern Transvaal Branch (M.A.S.A.)—*Editor.*]

St. Monica's Home

OBSTETRICAL HOUSE SURGEON

Applications are invited for the above-named position and should reach the Honorary Superintendent, St. Monica's Home, Lion Street, Cape Town, on or before 29 December 1951.

The successful applicant will commence duty early January 1952.

Required

Long-term assistantship (part-time or full-time) offered in Cape Town practice from February 1952. Apply to 'A. J. R.', P.O. Box 643, Cape Town, stating age, experience, etc.

Examination Couch Wanted

The Students' Health Service of the University of Cape Town, require an old examination couch. Please telephone 5-1356.

Transvaal Provincial Administration

VACANCIES : TRANSVAAL PUBLIC HOSPITALS

Applications are invited from suitably qualified candidates for the undermentioned posts at Public Hospitals in the Transvaal.

Applications should be addressed to the Medical Superintendents of the Hospitals concerned and should contain full particulars as to the age, professional, academic and language qualifications, experience and conjugal status of the applicant and should further indicate the earliest date upon which duties can be assumed. Copies only of recent testimonials to be attached.

Hospital	*Vacant Post*	*Emoluments*	*Remarks*
Boksburg-Benoni:	Full-time Assistant Physician	£1,150	Must be qualified for 5 years and must have at least 1 year's practical experience. Married plus (*a*) below. Single plus (*b*) below.
Pretoria:	Junior Assistant Radiologists (Itinerant) (2)	£1,200 x 50—1,500	Must be a registered medical practitioner. Plus a hardship allowance of £150 p.a. Married plus (*a*) below. Single plus (*b*) below.
	Junior Assistant Radiologists (1)	£1,200 x 50—1,500	Must be a registered medical practitioner. Married plus (*a*) below. Single plus (*b*) below.
	Senior Physician (1)	£2,000 p.a.	Must be a registered medical practitioner and must be suitably qualified through training and experience. Married plus (*a*) below. Single plus (*b*) below.
	Casualty Officer (1)	£620–780–820–860	Must be a registered medical practitioner. Married plus (*a*) below. Single plus (*b*) below.
Vereeniging:	Casualty Officer (1)	£600 p.a.	Married plus (*a*) and (*c*) below. Single plus (*b*) below.

(*a*) £256 per annum cost-of-living allowance.
(*b*) £80 per annum cost-of-living allowance.
(*c*) £60 per annum temporary allowance.

Full-time employees receive in addition to their salaries and cost-of-living allowance, the following privileges: Leave and rail concession.

Closing date of applications: 31 December 1951.

Application forms are obtainable from the Provincial Secretary, Hospital Services Department, P.O. Box 383, Pretoria. (32657)

National Industrial Council of the Leather Industry of South Africa

SICK BENEFIT FUND

Applications are invited from medical practitioners resident in the Magisterial area of Durban for appointment to a panel of part-time Medical Officers to the above Fund to commence duties early in the New Year.

Full details of conditions of appointment may be obtained from the Secretary, Sick Benefit Fund, National Industrial Council of the Leather Industry of South Africa, P.O. Box 3051, Port Elizabeth. Applications for appointment to the Fund should be similarly addressed and must be received not later than Monday, 14 January 1952. (LSF/5)

House To Let

Doctor's modern all electric seaside house to let for the holiday season. Situated Mosselriver, Hermanus. Apply to P.O. Box 42, Grabouw.

City of Durban

VACANCY FOR PART-TIME MEDICAL SPECIALIST (FEMALE)

Applications are invited from registered female medical specialists in obstetrics and gynaecology for the above-named part-time appointment in the Child Health Section of the City Health Department.

The duties appertaining to the position involve professional attendance at Municipal clinics in connexion with ante-natal clinical work. The provisional programme will require the successful applicant to attend the following Municipal clinic sessions:—

Gale Street: Every Thursday—2 p.m. to 3.30 p.m.

Brook Street: Every alternate Monday and Friday—10.30 a.m. to 12 noon.

The remuneration will be at the rate of £1 11s. 6d. per hour, payable monthly. Termination of duties will be subject to 3 calendar months' notice on either side.

Applications stating age, marital state, qualifications and experience and accompanied by copies of not more than 3 recent testimonials should reach the City Medical Officer of Health, Gale Street, Durban, not later than 12 noon, on Saturday, 5 January 1952.

Personal canvassing for appointment is prohibited and proof thereof will disqualify a candidate *vide* Council's standing order No. 1.

John McIntyre
Town Clerk

Town Clerk's Office
Durban
22 December 1951
(1502 C)

Town Council of Vereeniging

STAFF VACANCY : MEDICAL OFFICER OF HEALTH

Applications are hereby invited for appointment to the vacant position of Medical Officer of Health on the salary grade £1,200—100—£1,500 per annum, plus a temporary cost-of-living allowance which at present is £256 per annum for married persons and £78 per annum for single persons. A locomotion allowance is also paid, and the successful applicant will be required to provide his own car.

The successful applicant will be required to pass the medical examination as prescribed by the Council before assuming duties, and the appointment will be subject to the Council's general conditions of service as amended from time to time.

Applications must be submitted on the Council's official application form, which may be obtained, upon request, from the undersigned, and must reach the Town Clerk, Municipal Offices, Vereeniging, not later than 12 noon, on Friday, 18 January 1952.

Personal canvassing for appointment in the gift of the Council is strictly prohibited and proof thereof will disqualify a candidate.

I. T. McPherson
Town Clerk

Municipal Offices
Vereeniging
7 December 1951
No. 697
(B8722)

National Industrial Council of the Leather Industry of South Africa

SICK BENEFIT FUND

Applications are invited from medical practitioners resident in the Municipal area of Stellenbosch for the appointment as part-time Medical Officer to the Fund, to commence duties early in the New Year.

Full details as to conditions of appointment may be obtained from the Secretary, Sick Benefit Fund, National Industrial Council of the Leather Industry of South Africa, P.O. Box 3051, Port Elizabeth. Applications for appointment to the Fund should be similarly addressed and must be received not later than Monday, 14 January 1952. (LSF/5)

Required

Part-time medical officer required for factory in Boksburg North. For full particulars write to the General Manager, P.O. Box 63, Boksburg North.

Printed by Cape Times Ltd., Parow, and Published by the Proprietors, THE MEDICAL ASSOCIATION OF SOUTH AFRICA, MEDICAL HOUSE, 35 Wale Street, Cape Town. P.O. Box 643. Telephone 2-6177. Telegrams: 'Medical'

in Amœbiasis . . .

one out of six patients

showed no symptoms

but *all* 34 patients in this study carried Endamœba hystolytica[1] in their stools! Five were classified as asymptomatic and 18 were "persons with such poorly defined symptoms that they would not normally seek medical assistance . . . " but stool examination proved that all had amœbic dysentery.

In these instances, a course of treatment with Milibis and Aralen was completely successful. Milibis—bismuthglycolylarsanilate—a new intestinal amœbacide, is one of the most powerful of the drugs commonly used against Endamœba hystolytica.[2] Yet its toxicity is so low that side effects are virtually unobserved.

Aralen (chloroquine) diphosphate has been shown to exert a specific action on extra-intestinal amœbiasis. The combination of Aralen with a superior intestinal anti-amœbic drug such as Milibis furnishes adequate treatment of any amœbic infection.

HOW SUPPLIED:

Milibis, tablets of 0.25 Gm; bottles of 50;
Aralen, tablets of 0.25 Gm; tubes of 10 and bottles of 100.

***MILIBIS*—amœbacide . . . high potency . . . low in side effects**

***ARALEN*—diphosphate . . . for extra-intestinal amœbiasis**

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Winthrop Products
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Aralen, Milibis: regd. Trade-Marks.

[1] Towse, R.C.; Berberian, D.A.; and Dennis, E.W.: New York State Jour. Med.: 50:2035, Sept 1950
[2] Berberian, D.A.; Dennis, E.W.; and Pipkin, C.A.: Am. Jour. Trop. Med.: 30:613, Sept. 1950.

6486-I

Butesin Picrate Ointment with Metaphen

acquaint yourself with these facts about Butesin Picrate

1 Butesin Picrate combines picric acid with Butesin, an effective topical anesthetic and analgesic.

2 Butesin Picrate Ointment with Metaphen quickly relieves the pain of burns, scalds, ulcers, hemorrhoids, lacerations and abrasions; and acts as a soothing, protective, bacteriostatic dressing for the denuded area.

3 Butesin Picrate is accepted by the Council on Pharmacy and Chemistry of the American Medical Association.

4 Butesin Picrate Ointment with Metaphen is composed of 1 per cent. Butesin Picrate and Metaphen 1 : 5,000 incorporated in a vehicle composed of white wax, paraffin, petrolatum, water and sodium borate.

5 Butesin Picrate Ointment with Metaphen is available everywhere in prescription pharmacies, or may be ordered from the nearest Abbott branch or Home Office.

Available in tubes of 1oz. and 2oz. Also in 1-lb. tins, from

Abbott **Laboratories S.A. (Pty.) Ltd.**

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